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VOL. 30, NO. 5 NOVEMBER, 1958

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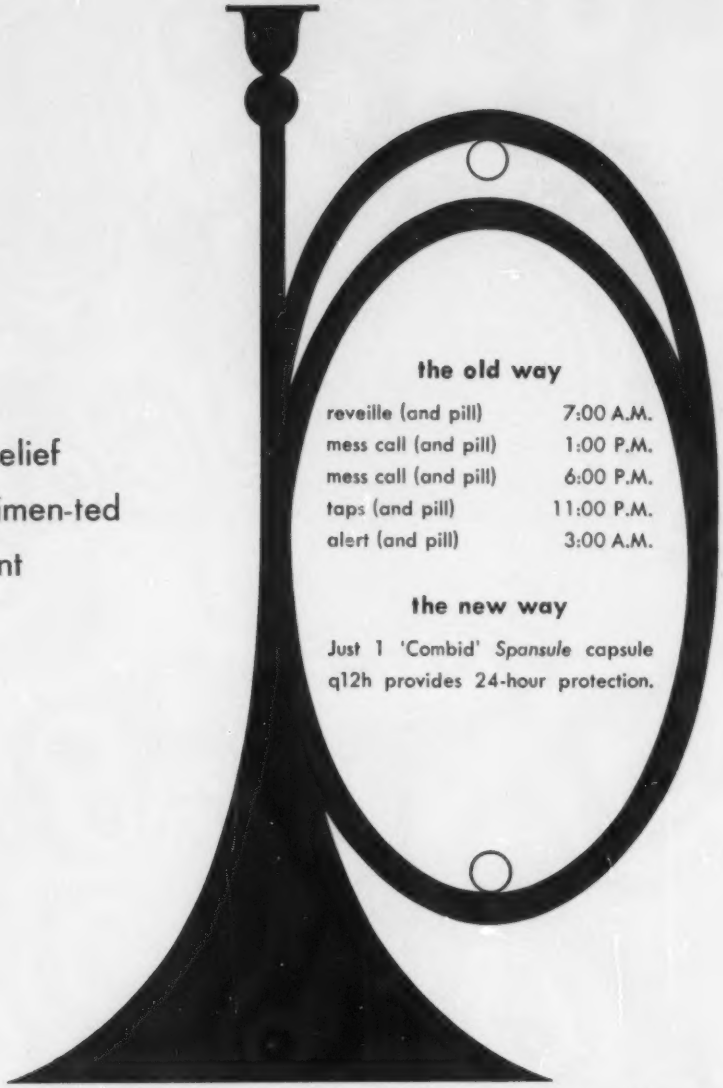

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THE American Journal OF Gastroenterology

(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

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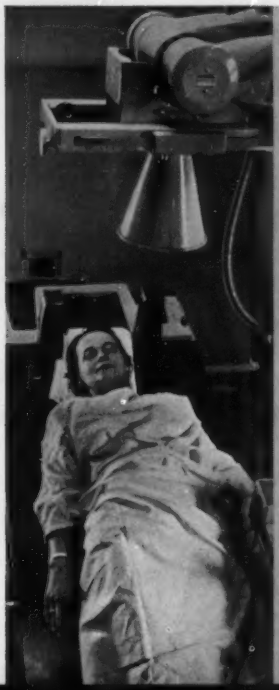

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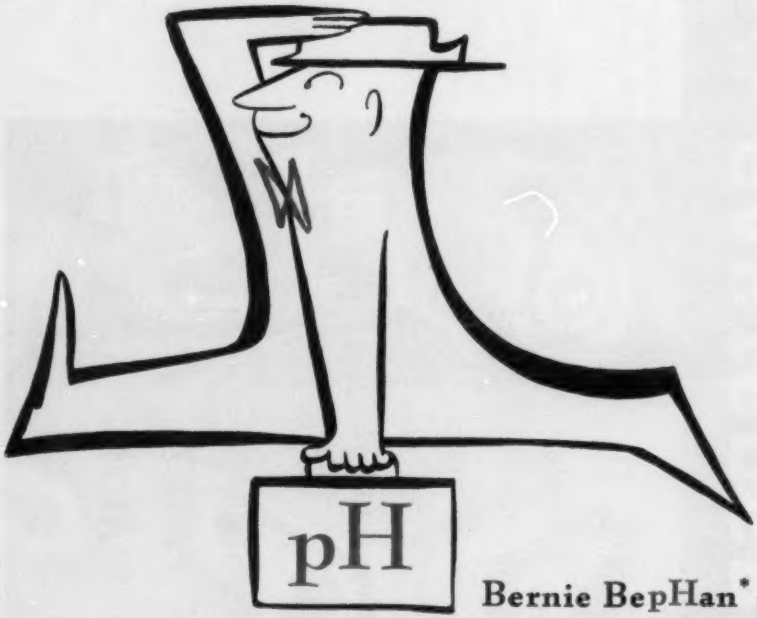

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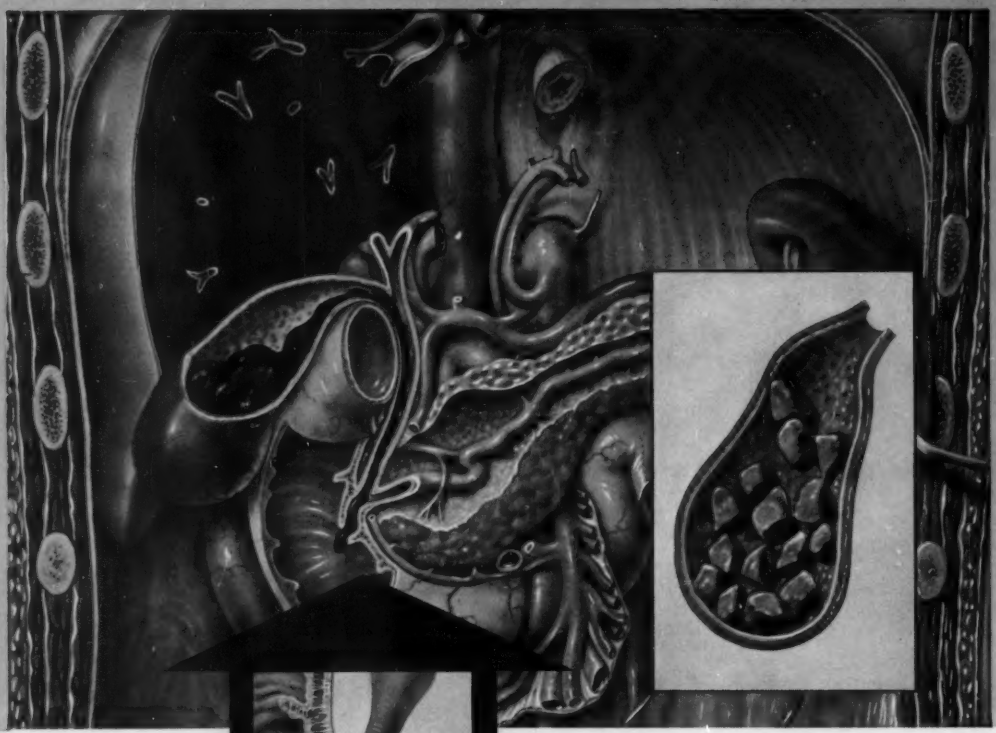

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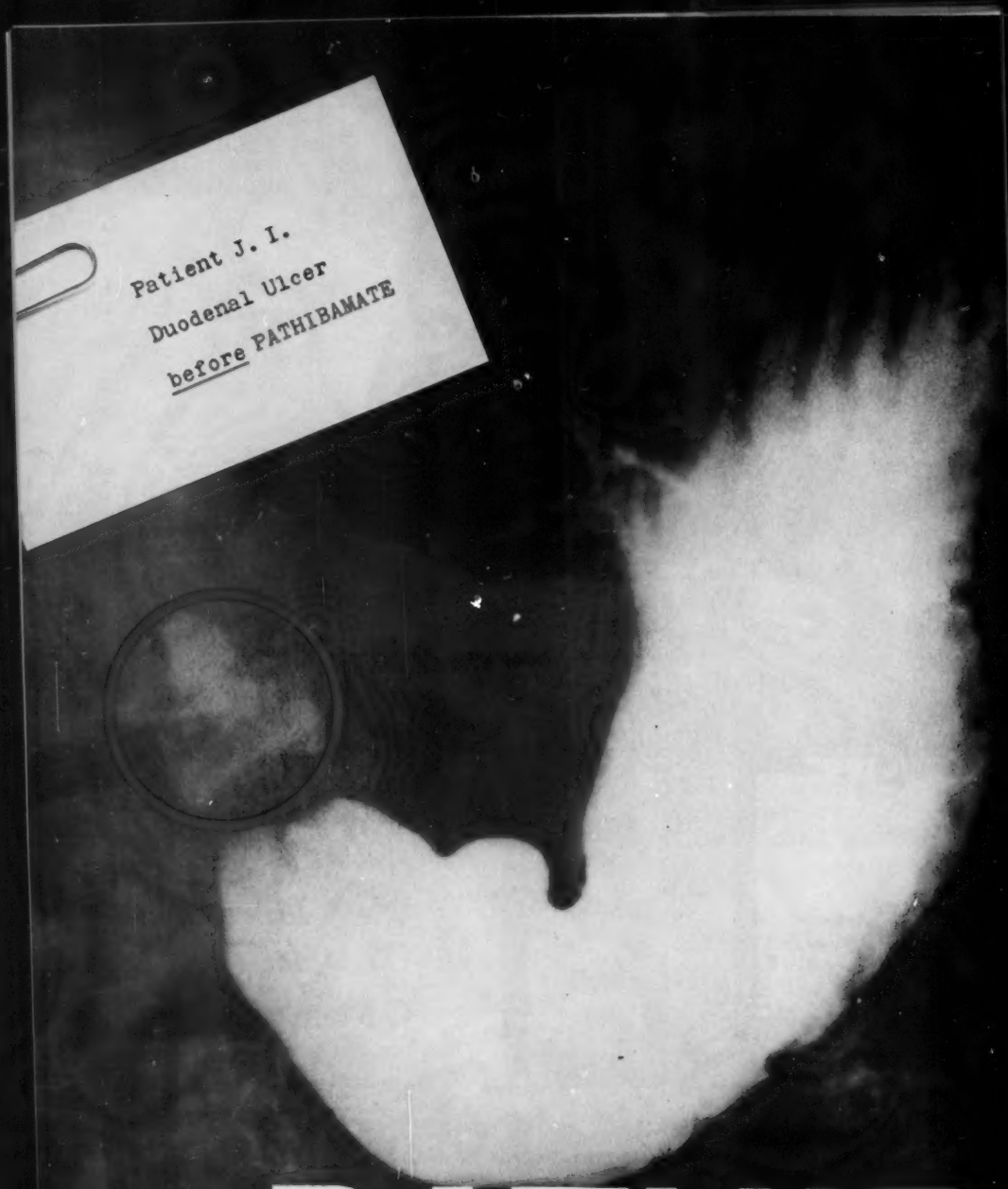

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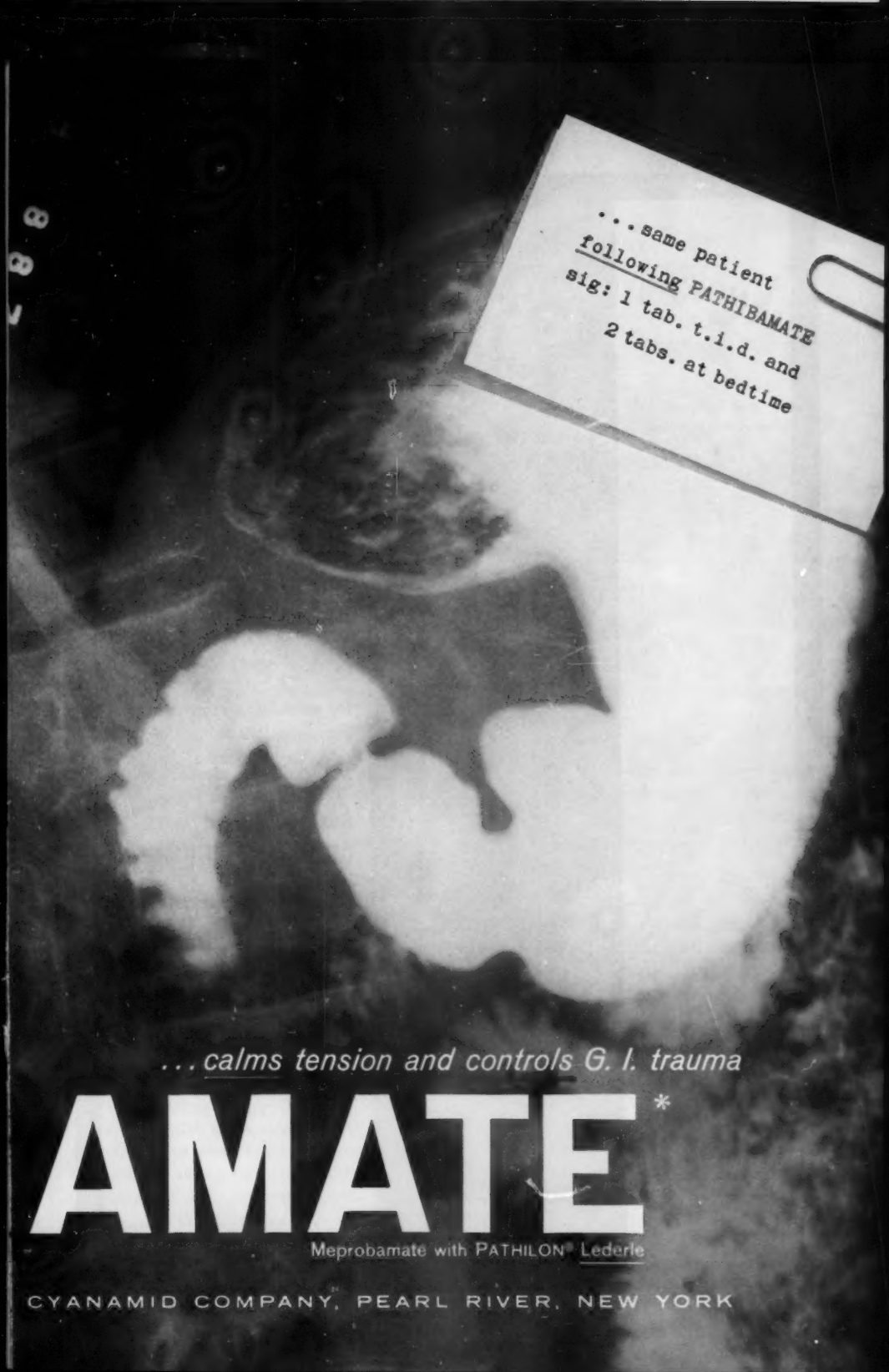

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OBSERVATIONS ON THE COURSE OF ACHALASIA TREATED WITH MECHANICAL (STARCK) DILATATION WITH SPECIAL REFERENCE TO REFLUX

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Englewood, N. J.

Any successful treatment of esophageal achalasia must not only satisfactorily relieve the patient's dysphagia but must maintain enough of the cardio-esophageal junction's function to prevent regurgitation with its attendant esophagitis. This report is a preliminary evaluation of how mechanical dilatation satisfies both criteria.

For many years forcible mechanical dilatation has been used in the treatment of cardiospasm. Olsen et al used hydrostatic dilatation on 452 patients. Satisfactory results were obtained in 69.2 per cent, but an additional 10.9 per cent might have been improved if they had been dilated again[1]. Allison used the hydrostatic dilator successfully in 46 of 49 patients[2]. Berdal and Gulli had "good" to "excellent" results with the Mosher dilator in 21 of 23 patients (91 per cent)[3].

Crump, Flood and Hennig, using the Starck dilator, reported in 1952 satisfactory results in 82 per cent of 72 patients after one dilatation and a total of 95 per cent good results after 2 to 3 dilatations. The follow-up period was 3 to 8 years[4]. Schindler reported complete success in 24 of 26 (92.3 per cent) patients using the same instrument[5]. In 1956 this series had increased to 80 cases and a 95 per cent success rate was noted; most of the patients were treated by one dilatation[6].

From the Medical Service of the Bronx Veterans Administration Hospital.

These results for the relief of dysphagia compare favorably with those obtained by the currently used surgical procedure—esophagocardiomyotomy (Heller). Hawthorne noted that all of his 35 cases had immediate relief of symptoms and that 94 per cent had lasting benefit in a follow-up period of 3 months to 7 years[7]. Barrett and Allison also found the procedure to be "successful" in all of their cases[2]. In Gammelgaard, Iversen and Thomsen's series of 31 cases 26 (84 per cent) had satisfactory benefits[8]. In the 7 cases in which Sweet performed the operation 6 had good relief of symptoms and the remaining patient had slight pyrosis[9]. Olsen, Ellis and Creamer had good to excellent results in 23 (85 per cent) of 27 cases followed long enough to make some sort of evaluation[10].

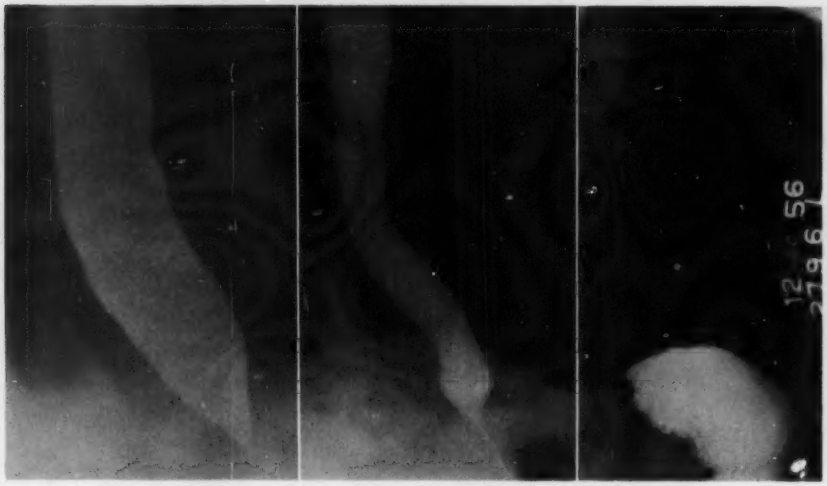

Fig. 1a Fig. 1b Fig. 1c

Fig. 1a—Pretreatment esophagram:—Note dilatation of esophagus with hold up at cardia.
Fig. 1b—Posttreatment esophagram:—Note some change in caliber of esophagus although there still appears to be a hold up at cardia.
Fig. 1c—Mueller procedure in Trendelenburg position fails to push barium into esophagus.

There is a relatively high incidence of peptic esophagitis following the other operative procedures which affect the integrity of the cardioesophageal junction. For some reason stricture rarely complicates the esophagitis although anemia from persistent occult, and occasionally overt, bleeding is not uncommon. Resection of the cardioesophageal junction, esophagogastrostomy (Heyrovsky), esophagogastrotomy with cardiotomy (Gröndahl) and cardioplasty (Wendel) have been the most common procedures. Olsen et al followed 11 cases from 4 months to 6½ years after esophagogastrostomy; 5 results were "poor" (2 died from hemorrhage related to esophagitis) and 1 was "fair"[1]. Six-

teen of the 19 patients in another series with esophagogastrostomy suffered from a postoperative peptic esophagitis[11]. Sweet noted that of 9 patients who had resections of the cardioesophageal junction 3 had severe and 1 had slight pyrosis[9].

The results following plastic repairs of the cardia are no better. Of the 4 cases followed up to 6 years by Olsen et al 2 were considered "fair" and 1 a "poor" result[1]. Kay noted an incidence of 28 per cent of reflux esophagitis in 32 patients who had cardioplasties[12]. Four of Barrett and Franklin's 6 cases of cardioplasty had this complication, although one was symptomatically better[2,11].

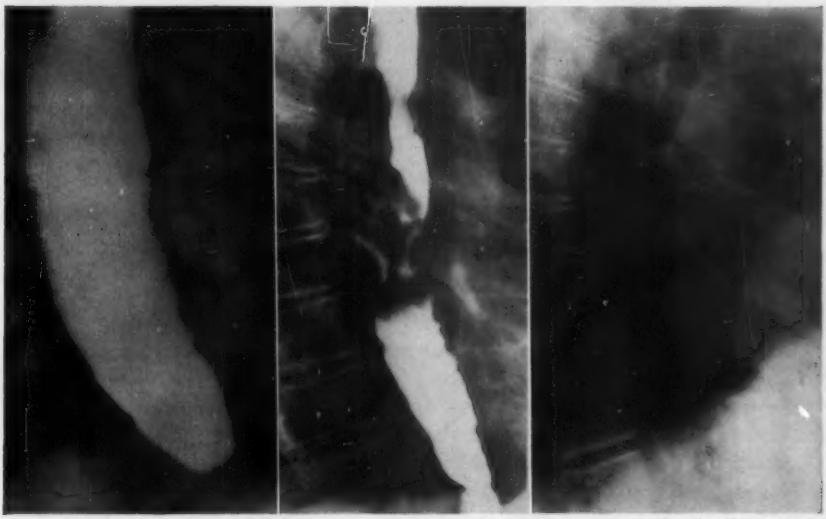

Fig. 2a Fig. 2b Fig. 2c

Fig. 2a—Pretreatment esophagram showing large dilated esophagus and a hold up of barium at cardia.

Fig. 2b—Posttreatment esophagram:—Note decrease in size of esophagus, presence of tertiary contraction waves, and potency of cardia.

Fig. 2c—Mueller maneuver in Trendelenburg position:—Note barium in stomach but no reflux into esophagus.

Five of Sweet's 20 cardioplasty patients complained of regurgitation; 4 other cases bled from gastritis and 1 bled from gastritis and esophagitis[9].

Esophagocardiomyotomy has had a considerably better record concerning peptic esophagitis. Sweet followed 7 patients one of whom had slight pyrosis and another had x-ray evidence of esophageal spasm. None had regurgitation either subjectively or by x-ray[9]. Of the 10 cases reported by Bugden and Delmonico none had "serious postoperative complaints or complications"[7]. Barrett followed 11 cases after the Heller operation and found no radiographic

evidence of reflux with the patient in the Trendelenburg position and "neither acid nor pepsin in the gullet"[2]. In 1956 Nemir et al reported 35 cases followed for 4 months to 6 years after the Heller procedure. They showed by x-ray that reflux was readily evident in 50 per cent[14,15]. All of the 21 per cent poor results of the whole series had reflux[7]. Of the 31 cases followed by Gammelgaard, Iversen and Thomsen no evidence of reflux was noted with the patients in the Trendelenburg position—however the Valsalva or Mueller maneuvers were not used. Fifteen (49 per cent) of the patients had no complaints, 11 (35 per cent) had occasional retrosternal oppression and regurgitation[8]. All the unsatisfactory results were thought to be due to concomitant disorders or to technical faults in the procedure. Thus it appears that although reflux is less frequent following the Heller procedure it still occurs.

Method of Study

1. Six patients who had had mechanical (Starck) dilatation of the cardia for achalasia were studied.

2. The patients' symptoms were reevaluated and esophagrams were done.

3. An obturator-stomach tube (34F Crump) was passed into the esophagus; the duration of delay at the region of the cardia was timed and the "feel" of the hold-up noted.

4. Under fluoroscopic control 240 ml. of barium sulfate-water suspension was injected through the Crump tube which was then withdrawn. Sixty ml. of water was then given by mouth to wash down any barium that might have dripped from the tube. The patient as quickly placed on the tilt table, turned to the supine position, tipped to 20° Trendelenburg and fluoroscoped at which time the Meuller maneuver was performed. In no instance was the barium suspension noted to reflux from the stomach. Radiographs were then taken during the Meuller maneuver without moving the patient off the table or changing his position.

Results

The follow-up periods ranged from 6 weeks to 42 months. The duration of symptoms before the procedure was from 6 months to 19 years. Most of the patients had had previous medical treatment with varying degrees of success. Weight loss varied from none to 30 pounds (Table I).

Following dilatation improvement was good to excellent (no dysphagia, vomiting or regurgitation, little or no pyrosis and a significant weight gain) in 5 patients. Results were characterized by the patients with such remarks as "can eat anything", "99 per cent improvement", "almost 100 per cent improvement" and "75 to 80 per cent improvement". The results were apparent very soon after the procedure; usually the following day when the patient was allowed his first

TABLE I

SUMMARY OF RESULTS

Pt.	Age	Duration sympts. before dilatation	Duration of F.U.	Result	Wt. gain (lbs.)	Duration tube hold-up	Esophagram
1	42	12 years	42 mos.	Good. Able to eat solids; occ. "sticking" when under stress. No pyrosis or regurgitation.	40	16 sec.	Moderate distention with tertiary waves. No reflux.
2	47	5 years	26 mos.	Excellent. Eats solids without trouble. No pyrosis or regurgitation.	10	1 sec.	Moderate distention with some change after procedure; tertiary waves. No reflux.
3	47	19 years	10 mos.	Fair. "90% improved" for 1st mo. then sympt. about ⅓ as much as before dilatation. Slight pyrosis. No regurgitation.	0	"Slight"	No distention before or after procedure. No reflux.
4	64	18 years	16 mos.	Excellent. Some pyrosis up to 1 year but no pyrosis for last 5 mos.; no regurgitation.	20	None	Moderate distention, about ⅓ as much as before procedure; marked tertiary waves. No reflux.
5	35	6 years	5 mos.	Fair. Good initial result, then some recurrence. After second dilatation: Excellent, able to eat "everything". No pyrosis or regurgitation.	2 14	8 sec. 0	Marked distention. Little change in procedure. No reflux.
6	22	8 months	6 wks.	Excellent, eats everything except raw apples and pears. No pyrosis or regurgitation.	30	30 sec.	Marked distention; no change in procedure. Questionable widening at vestibule. No reflux. Positive Mecholyl test.

meal. Weight gains ranged from none (in the patient who had lost no weight) to 40 pounds. None of the patients had regurgitation (either when supine or when bending forward), nor vomiting or signs of overt gastrointestinal bleeding. Four had no pyrosis and one complained of slight heartburn two or three times a month for about one year after the procedure but none for four months when last seen. One patient required two dilatations because of only a fair result after the first instrumentation. The second dilatation done two and a half months later yielded an excellent result.

The patient who was not improved to a gratifying degree had a good result for the first month after dilatation but then noted some recurrence of the symptoms. He noted no regurgitation but a sour burning taste in the throat when supine. He had slight pyrosis, but to a lesser extent than before the dilatation. This was more prominent while erect. No weight loss was encountered before the procedure and no weight was gained afterwards. This patient was the most apprehensive of the entire group and had the least amount of esophageal distention. He may well have had a diffuse esophageal spasm which played a greater role than the spasm of the vestibular region.

The hold-up of the obturator-stomach tube at the cardia did not seem to correlate with the clinical success of the dilatation. A patient with a "good" ("75 to 80 per cent improvement") result had a 16 second hold-up whereas the man with the poorest result had a delay of only a few seconds on the first passage of the tube and none on a subsequent intubation.

The degree of distention of the esophagus as seen on the posttreatment radiographs correlated poorly with the amount of improvement. The case with the least satisfactory result had the smallest amount of distention and one of the "excellent" results had little change in the large esophageal diameter after the dilatation. In others the esophagus was apparently narrower but this may have been due to the lesser degree of filling because of more rapid egress of the contrast material into the stomach.

Case Reports

Case 1:—A 42-year old male postal clerk had a 12-year history of dysphagia with substernal pressure after eating a small amount of food and vomiting afterwards. He did fairly well, gaining 10 pounds, on a regimen which included antispasmodics and bouginage with a 46F mercury weighted Hurst bougie over the next 5 years. Because of the continued symptoms he was admitted to the hospital and on 17 September 1953 dilatation with the Starck instrument was accomplished.

The patient was then able to eat solid foods and rapidly gained 40 pounds. He considered himself "75-80 per cent" improved, suffering some mild recurrence of the symptoms occasionally (especially when under stress). He has had

no pyrosis, regurgitation, or gastrointestinal bleeding. Passage of the obturator-stomach tube three years later encountered an 18 second hold-up. No reflux was found radiographically. Moderate esophageal distention was noted with numerous tertiary contractions. The predilatation x-rays were lost making comparison of esophageal size impossible.

Case 2:—A 47-year old porter had a history of intermittent dysphagia for 4 years prior to dilatation. He was admitted to the hospital late in 1954 for progressive dysphagia and a 10 lb. weight loss. After nightly esophageal aspirations for 3 weeks the Starck dilator was used on 6 January 1955.

He had marked improvement, considering this to be "almost 100 per cent". He was able to ingest solid food without any dysphagia. A weight gain of about 10 pounds ensued in three weeks. No pyrosis, regurgitation or gastrointestinal bleeding were noted. Passage of the obturator-stomach tube, over 1½ years later, encountered a delay of 1 second at the cardia. No reflux was found by x-ray examination. There was a definite decrease in the caliber of the esophagus in the postdilatation films; a considerable number of tertiary contractions were seen (Fig. 1).

Case 3:—A 47-year old male clerk with a 19-year history of dysphagia had been treated intermittently at another hospital with transient benefit. Frequent episodes of vomiting with blood-streaking and pyrosis at the level of the xiphoid were the main complaints at the time of admission. On 19 April 1956 dilatation with the Starck instrument was performed.

For the first month after the procedure marked improvement was reported but thereafter the good result deteriorated into only a fair one; there was no regurgitation but burning in the mouth when supine and some pyrosis (when erect) were noted. There was slight blood-streaking of vomitus on one or two occasions over the 10 month period of follow-up after the dilatation. Slight delay of the obturator-stomach tube was noted at the cardia. No reflux was seen by x-ray; the esophagus was normal in width and not very different from appearance from the predilatation study. The patient was quite apprehensive and not especially cooperative. Although advised to return for further evaluation and treatment, he failed to do so. There is reason to believe that this may not represent typical achalasia but rather a more diffuse esophageal spasm.

Case 4:—A 64-year old male had an 18-year history of regurgitation 3-4 times a week. The diagnosis of achalasia was made 8 years earlier. Because of increasing symptoms and a 7 pound weight loss he was hospitalized in 1955. On 6 October 1955 the Starck dilator was used and marked resistance noted to dilatation.

Gratifying results were soon apparent, dysphagia and regurgitation disappearing. A 20 pound weight gain ensued and the patient regarded himself as "99 per cent improved". He had several short episodes of slight pyrosis up

to 1 year after the procedure, but none since. No significant obstruction to the obturator-stomach tube was encountered. There was no radiographic evidence of reflux. A considerable decrease in the width of the esophagus was found after the dilatation (about ⅓ of the previous diameter); numerous tertiary contractions were seen (Fig. 2).

Case 5:—A 35-year old male elevator operator had a 6-year history of dysphagia. He was hospitalized in 1950 because of 3½ months of frequent vomiting and a 40 pound weight loss. Achalasia was diagnosed and he was treated with the Hurst dilator and did fairly well regaining some weight. He was readmitted because of a recurrence of the symptoms. Dilatation with the Starck instrument was performed against moderate resistance on 2 August 1956.

He had a fair result initially good but then deteriorating. A weight gain of only 2 pounds was noted. The obturator-stomach tube was held up 8 to 25 seconds at the cardia.

He was readmitted and dilated again on 11 October 1956. Following this he did well, stating he was able to eat anything and felt "like new". A 16 pound gain occurred. No pyrosis, regurgitation or signs of gastrointestinal bleeding were noted. (Reflux was not found by x-ray). No significant hold-up of the obturator tube was found. There was little change in the caliber of the esophagus.

Case 6:—A 22-year old male college student was admitted with an 8-month history of intermittent vomiting, dysphagia, and a 40 pound weight loss. Regurgitation sometimes of food from the previous day, was noted when in the recumbent position. A distressing nocturnal cough developed.

A good immediate result was obtained and the day following dilatation the patient noted that fluids descended into the stomach promptly. He found that he was able to swallow everything except raw apples and pears. A 30 pound gain occurred in 6 weeks. He felt he had had a "90 per cent improvement". No pyrosis, regurgitation or signs of bleeding were encountered. A hold-up of 30 seconds at the cardia was noted with the obturator-stomach tube. No reflux was found on the x-rays. There was no significant change in esophageal size and some equivocal widening in the region of the vestibule was seen. A Mecholyl test, using 10 mg. i.m., was positive 3-4 minutes after the injection of the drug.

Comment

As far as could be determined, no other study has been reported where cases of achalasia treated by mechanical dilatation were systematically examined to ascertain the presence of esophageal reflux. Schindler, following 84 cases, mentioned that "no peptic esophagitis develops"[6]. Berdal and Gulli did did not observe any regurgitation of peptic esophagitis in the 20 cases dilated with the Mosher pneumostatic apparatus and followed from 1 to 48 months[3]. None of our 6 cases had regurgitation, subjectively or radiographically.

In achalasia the "failure of relaxation" of the vestibule and/or inferior esophageal constrictor along with the concomitant dysrhythmia of the more proximal esophagus are the primary features[16, 17]. All the surgical procedures, with the exception of the Heller operation, either destroy or by-pass the sphincters or valves and disturb the relationship of the esophagus to the diaphragm. The Heller operation apparently largely interferes with the "valve" and/or "sphincter" actions and probably does not significantly alter the esophagophrenic relationship nor interrupt the muscle bands going over the fundus which may play a role in the "closing mechanism"[8]. This may explain the lower incidence of reflux. It seems highly probable that esophagocardiomyotomy involves a much greater length of muscle interruption than dilation with the Starck instrument since the "blades" are only 6 cm. long and during dilatation only part of the length is in the esophagus. The end result, however, may be the same after either procedure with less reflux after dilatation.

It would seem at present that forcible dilatation (preferably with the Starck instrument because the operator can "feel" the degree of resistance without relying on pressure gauges) yields symptomatic relief in achalasia as adequately and as frequently as esophagocardiomyotomy. The morbidity, when used in experienced hands, is less than with surgery and the long-term complications so far would seem to be less. Contrary to the oft-stated opinion frequent dilatations are not needed, the majority of cases responding to one manipulation and almost all cases to two or three. The few "undilatable" cases can be subjected to the Heller procedure.

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PRANTAL WITH PHENOBARBITAL OR WITH TRILAFON IN GASTROINTESTINAL DISEASES*

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Recognition and treatment of emotional problems is a *sine qua non* of adequate management of patients with peptic ulcer and similar gastrointestinal diseases[1,2]. Manipulation of the environment or psychotherapy to remove these factors at their source is desirable but frequently impossible; therefore, symptomatic treatment of tension and anxiety with sedatives or tranquilizers is accepted practice. We usually prescribe an anticholinergic agent with phenobarbital along with standard measures such as bland diet, frequent milk feedings, and antacids. Prantal (diphemanil methylsulfate), a quaternary ammonium compound with marked anticholinergic activity, available alone or with phenobarbital, has been used successfully[3,4].

The tranquilizers, particularly those derived from phenothiazine, are used frequently in peptic ulcer. They are relatively nonhabituating and generally do not provoke an undesirable amount of sedation among ambulatory patients. Favorable results with Trilafon (perphenazine) have been reported in patients with gastrointestinal disease complicated by a significant anxiety component[5,6].

Method

The series consisted of 48 patients with various gastrointestinal diseases seen in private practice or in clinic at the Jersey City Medical Center. Duodenal ulcers were present in 38 patients, gastric ulcers in 4, hypertrophic gastritis with hyperacidity in 4, and esophagitis in 2. Diagnoses were confirmed by radiography, and gastroscopic and esophagoscopic examinations were performed when indicated.

All patients were placed on bland diets and antacids were administered. We wished to determine the relative effectiveness of Prantal alone, Prantal and Trilafon, or Prantal with phenobarbital‡. Originally, half of the patients received 100 mg. Prantal 4 times daily, and the other half received the same dosage of Prantal and also 2 or 4 mg. Trilafon 4 times daily. Those patients who did not respond to Prantal and Trilafon received, instead, 2 tablets containing 100 mg.

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‡These drugs were provided by Dr. G. Kenneth Hawkins, Division of Clinical Research, Schering Corporation, Bloomfield, N. J.

Prantal and 16 mg. phenobarbital 4 times daily for a few days and then this dosage was halved.

Results

A cure was considered to have occurred if there was relief of pain, heartburn, and regurgitation, if there was a gain in weight with marked general improvement in physical and emotional health, and if radiographic examination showed healing of the ulcer. According to these criteria all but 2 of our patients (both with duodenal ulcers) demonstrated satisfactory evidence of recovery. Complete healing usually occurred within 6 weeks to 3 months after therapy was begun.

About 75 per cent of the patients who received Prantal and Trilafon were later placed on Prantal with phenobarbital with continued improvement.

There were no side-effects other than drowsiness among a few patients who received 16 mg. Trilafon daily.

The following case reports are typical of those in our series of patients who were successfully treated with Prantal and Trilafon:

Case 1:—A female, 67 years old, extremely nervous, complained of burning retrosternal and epigastric pain through to the back, difficulty in swallowing, and moderate weight loss, over a period of 2 years. The diagnosis was esophagitis, hypertrophic gastritis, and hyperacidity. A bland diet with frequent milk feedings, antacids, and 4 mg. Trilafon with 100 mg. Prantal 4 times daily was prescribed. During 6 months of this therapy the patient's appetite improved, she gained weight, and symptoms receded. There were no side-effects from either drug.

Case 2:—A male, 45 years old, presented classical symptoms of a duodenal ulcer which was confirmed by radiographic examination. A bland diet with frequent milk feedings and antacids, and 4 mg. Trilafon with 100 mg. Prantal 4 times daily, was prescribed. Within 1 week the patient was completely relieved of pain. The medication was continued for an additional 3 weeks. No side-effects occurred.

Case 3:—A male, 24 years old, complained of severe pain in the upper abdomen radiating to the left side and the back. Radiographic examination revealed a small ulcer crater at the angle of the lesser curvature of the stomach. A bland diet with frequent milk feedings and administration of antacids was prescribed, along with 4 mg. Trilafon and 200 mg. Prantal 4 times daily. Within 3 weeks complete healing of the ulcer occurred and pain disappeared. There were no side-effects. The patient was maintained on 2 mg. Trilafon and 100 mg. Prantal daily for an additional 3 weeks.

Summary and Conclusions

Forty-eight patients with various gastrointestinal diseases were treated with Prantal (diphemanil methylsulfate) alone or in combination with phenobarbital or Trilafon (perphenazine). Radiographic and clinical evidence of healing occurred in all but 2 patients within 6 weeks to 3 months. Most patients required the combination of Prantal with phenobarbital for maximal improvement.

There were no side-effects except drowsiness induced in a few patients by Trilafon. It appeared to us that side-effects following Trilafon administration are markedly less frequent and less severe than those which we have encountered with other phenothiazine derivatives.

The combination of Prantal and Trilafon is useful in the treatment of peptic ulcer, hypertrophic gastritis and hyperacidity, and esophagitis. This regimen is more effective than Prantal alone. In some patients, Prantal with phenobarbital seemed to give superior results. The antispasmodic action of phenobarbital supplements that of Prantal and therefore this combination is indicated when a relatively strong drug effect is required. Prantal with Trilafon is useful in cases where there is a marked emotional component and in patients who tolerate phenobarbital poorly.

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OBSTRUCTIVE JAUNDICE SYNDROME AS A PHASE OF EXTENSIVE PARENCHYMATOUS LIVER DAMAGE DUE TO CIRRHOSIS*

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Introduction

In the presence of extrahepatic obstruction of long duration the differential diagnosis of jaundice arising from hepatocellular damage is difficult. This is particularly true when the history is nebulous and there are no prominent physical findings. The reverse can also be difficult, i.e., in some stages of cirrhosis, jaundice can present a picture which is difficult to differentiate by laboratory tests from jaundice due to acute extrahepatic biliary obstruction.

Phillips and Davidson[1] recently reported the cases of four cirrhotics in whom the possibility of obstructive jaundice was considered, two of whom were operated on. They recommended the use of liver biopsy by the Silverman needle technic as an aid in making the diagnosis. Although this aided in the diagnosis in Case 3, we have hesitated to utilize blind liver biopsy in the presence of possible extrahepatic biliary obstruction because of the dangers of bile peritonitis[2] or intraabdominal hemorrhage[3].

Sherlock[4] recognized the difficulty in diagnosis in patients with cirrhosis. She found that a correct initial clinical diagnosis was made in only 25 of 50 cases of jaundice due to cirrhosis. Biochemical data aided in the diagnosis of only ten further cases. In contrast, in 46 of 50 cases of acute hepatitis, and in 43 of 50 patients with obstructive jaundice, the admitting diagnosis was correct, with laboratory data enabling diagnosis of the majority of the rest.

We have recently encountered four cases of cirrhosis where the diagnosis of biliary obstruction was seriously considered. In these cases a complex of laboratory tests pointed predominantly to biliary obstruction and the history and physical findings were equivocal. Surgery was considered in three cases and laparotomy was actually carried out in one case. The diagnosis of cirrhosis was subsequently verified histologically at autopsy, and by surgical biopsy at the time of portacaval shunt for portal hypertension.

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Case Reports

Case 1:–E. B., a 51-year old white male was admitted on 15 September 1957 with a history of painless jaundice for several weeks, associated with anorexia, weight loss, acholic stools and dark urine. The patient called himself a "social drinker" and denied drinking to excess, previous jaundice, or exposure to hepatotoxins or injections. On physical examination several *spider angiomata* were present. He was deeply jaundiced and the liver was palpable three finger-breadths below the right costal margin.

A gastrointestinal series was inconclusive, but suggested an impression at the posterior wall of the stomach.

Laboratory results were as follows: Bilirubin, 13.4 (direct 8.74, indirect 4.65); Hanger, 2 plus; thymol turbidity, 2.5; alkaline phosphatase, 24.5; urine urobilinogen, 3.2 units; A/G, 3.5/4.2.

TABLE I

Summary of Liver Function Studies Suggesting Extrahepatic Obstruction

	Case 1	Case 2	Case 3	Case 4
Bilirubin (Total)	13.4	21.5	16.6	9.2
Dir./Indir.	8.74/4.65	9.15/12.4	—	—
Hanger	2	0	0	1
Thymol turbidity	2.5	5.3	5.0	2.5
Alk. Phosphatase	24.5	17.8	7.7	11.8
Urine Urobilinogen	3.20	3.08	2.50	—
A/G Ratio	3.5/4.2	3.6/3.4	3.7/2.7	2.7/2.3

Initially it was felt that this was hepatitis and he was discharged and followed at home. The jaundice persisted and he was readmitted on 28 October 1957. Physical examination was essentially the same except for emaciation and excoriation of the skin due to pruritus.

On this admission laboratory results were as follows: Bilirubin, 16; Hanger, 4 plus and then 2 plus on a repeat examination; thymol turbidity, 5; alkaline phosphatase, 30.2; urine urobilinogen, 1.56 units; A/G, 3.1/3.7.

At this time the persistent jaundice, lack of a history of excessive alcoholic intake, markedly elevated alkaline phosphatase, elevated direct bilirubin, inconsistently elevated Hanger, and normal thymol turbidity made an obstructive type jaundice seem most likely in spite of cutaneous stigmata of cirrhosis, and an elevated urine urobilinogen. The patient was explored on 5 November 1957 and no intrinsic biliary tract disease found. Liver biopsy revealed portal cir-

rhosis with degeneration and cholestasis. Postoperatively he went downhill rapidly and died on 13 November 1957. The poor response of the decompensated cirrhotic to the stress of operation points vividly to the importance of accurate differential diagnosis in this syndrome.

Case 2:—J. S., a 52-year old white male was admitted on 18 November 1957 after he had noticed jaundice and dark urine two days earlier. He had previously had a subtotal gastrectomy for a massive upper gastrointestinal hemorrhage, secondary to duodenal ulcer, on 1 March 1956. He had drunk four to five drinks a day for 30 years. He denied previous jaundice, exposure to hepatotoxins or injections. On physical examination he was markedly jaundiced with several *spider angiomata*. A palpable mass thought to be liver was four fingerbreadths below the right costal margin.

Laboratory results were as follows: Bilirubin, 21.5 (direct 9.15, indirect 12.4); Hanger negative on three occasions; thymol turbidity, 7.3, 5.3 and 2.5; alkaline phosphatase, 17.8 and 12.4; urine urobilinogen, 3.08; A/G, 3.6/3.4.

At this time surgery was strongly considered in view of the laboratory findings which were strongly suggestive of obstructive jaundice. The patient failed rapidly and it was felt that the rapid deterioration bespoke of more hepatic damage than would be expected in the presence of pure obstruction of this short a duration.

The patient ran a persistent low grade fever, gradually became stuporous with an elevation of the serum ammonia to 135 gamma per cent (control 50 gamma per cent) and died on 25 November 1957. At autopsy microscopic examination of the liver showed a portal cirrhosis with subacute hepatitis. The biliary tree was free of disease.

Case 3:—M. K., a 43-year old white male was admitted on 20 February 1957 with a three-day history of jaundice and lethargy. He had drunk six to eight glasses of beer a day for a number of years. He denied exposure to hepatotoxins or injections. He had been jaundiced previously in 1943, while on Saipan. On physical examination he was deeply jaundiced, with several *spider angiomata*. There was a palpable mass thought to be liver five fingerbreadths below the right costal margin. A gastrointestinal series showed an enlarged ampulla of Vater.

Laboratory results were as follows: Bilirubin, 16.6; Hanger, plus-minus; thymol turbidity, 5; alkaline phosphatase, 7.7; urine urobilinogen, 2.5; A/G, 3.7/2.7.

This man had several physical signs compatible with cirrhosis, but his Hanger and thymol turbidity remained normal, and he had x-ray evidence suggestive of a neoplasm of the ampulla of Vater. A liver biopsy was performed on 5 March 1957, and read as portal cirrhosis and cholestasis. The patient

signed himself out of the hospital, but returned on 18 March with an upper gastrointestinal hemorrhage eventually controlled by Sengstaken balloon tamponade. A repeat gastrointestinal series failed to demonstrate the enlarged ampulla of Vater and splenoportography showed an intrahepatic type block with extensive collateralization. One month after the second admission his Hanger first became 4 plus. A portacaval shunt was performed on 20 June 1957. Coincidental inspection of the biliary tree revealed no extrahepatic obstruction. The patient has done well since.

Case 4:—J. C., a 54-year old white male was admitted on 19 November 1957 with a two-day history of hematemesis and diarrhea, and a three-week history of fatigue. Past history included a gastroenterostomy for a duodenal ulcer in 1929, and a thoracic vagotomy in 1950. He had had postprandial vomiting, right upper quadrant gas pains, bloating and belching for two years. The patient admitted a moderate alcoholic intake. He denied previous jaundice, recent injections, or exposure to hepatotoxins. On physical examination he had scleral icterus, a "doughy abdomen" with moderate upper abdominal tenderness, a questionable mass in the midepigastrium, pitting ankle edema, and tarry stool on rectal examination.

Laboratory results were as follows: Hemoglobin, 7 gm.; bilirubin, 9.2; Hanger, 1 plus (on two occasions); thymol turbidity, 2.5; alkaline phosphatase, 11.8; A/G, 2.7/2.3; amylase, 243.

He was transfused and the bleeding ceased spontaneously, but he gradually became comatose and expired on 1 December 1957.

This patient had laboratory data compatible with an obstructive type jaundice, an upper abdominal mass and tenderness, and a history of right upper quadrant pain. It was felt that a pancreatic tumor or choledocholithiasis with a concomitant bleeding duodenal ulcer was a good possibility, but he failed to stabilize sufficiently to consider exploration prior to his demise. At autopsy an active duodenal ulcer, and a nonfunctioning gastroenterostomy were found. The liver was enlarged and microscopic examination showed portal cirrhosis with fatty metamorphosis and necrosis. No extrahepatic blockage of the biliary tree, or pancreatic tumor was present.

Comment

At a stage in many cirrhotics, regardless of etiology, there can occur jaundice in which the history, physical examination, and particularly laboratory data, are indicative of an obstructive etiology. In our series the Hanger and thymol turbidity were indeterminate in all four cases, and the alkaline phosphatase was markedly elevated in three cases. This may lead to surgery, as in Case 1, and as contemplated in Cases 2 and 3. Urobilinogen in all three cases was elevated. This may be interpreted as suggestive of nonobstructive jaundice.

In the early stages of obstructive jaundice, however, it is not unusual to see these elevations.

Evaluation of the whole clinical picture led to the avoidance of the pitfalls that absolute reliance on laboratory procedures might have led to. One clinical observation was important in this respect, namely, that the patients all appeared extremely ill, much more so than would be expected in the presence of obstructive jaundice of short duration. In Case 1, this did not obtain since the patient had been ill and jaundiced for a long period of time and his poor condition could have been explained on the basis of a long standing extrahepatic biliary obstruction.

Summary

Four cases of cirrhosis, proved at postmortem or operation, who exhibited a picture of obstructive jaundice during their courses, are described.

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D-GLUCITOL IN THE TREATMENT OF BILIARY TRACT DISEASE

Preliminary Observations

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D-glucitol, a hexahydric sugar alcohol derived from the catalytic dehydrogenation of glucose, has at various times in its past been used as a sweetening agent and a nonhyperglycemic carbohydrate for diabetics[3]. During the screening of its metabolic properties, Sabatini and Gigante[1] inadvertently discovered that it also effected contraction of the gallbladder. One year later Seitz[2], after doing introduodenal volume and pressure studies, concluded that it was a synchronous relaxant of the sphincter of Oddi as well. In addition, he found that although it was effective orally, it was ineffective intravenously, and further concluded that its cholecystosphincteric action was mediated through a gastrointestinal reflex. Since then Wissner[3], Caroli[4], and Piccinelli and Timossi[5] have exploited the cholecystosphincteric effect of d-glucitol with good results in the treatment of acute and chronic cholecystitis, functional biliary dyskinesia, the postcholecystectomy syndrome, and cholelithiasis.

TABLE I

Clinical Response

Disease	0	1	2	3	Total
Chronic cholecystitis	0	2	10	5	17
Postcholecystectomy syndrome	0	0	5	4	9
Delayed emptying	0	0	2	5	7
Cholelithiasis	0	3	2	0	5

This report is a preliminary study of the therapeutic effects of a preparation* containing d-glucitol and the antispasmodic, diisopromine (N,N[1] diisopropyl-3, 3-diphenylpropylamine), in selected cases of biliary tract disease.

Materials and Methods

There were 38 cases studied, including 17 cases of chronic cholecystitis with no proven evidence of stones, 9 of the postcholecystectomy syndrome, 7 of delayed emptying of the gallbladder with no other evidence of disease, and

*IsoBilagol, supplied by the Purdue Frederick Company, New York, N. Y.

5 of cholelithiasis. They ranged in age from 29 to 74. Of the 38, 28 were females and 10 were males. All were symptomatic: the symptoms observed were fat intolerance, abdominal discomfort, nausea, eructations, pain, and constipation.

The preparation contains 4.5 gm. of d-glucitol and 2.0 mg. of diisopromine per 5 ml. (1 teaspoonful); the diisopromine is used to reenforce the relaxant action of d-glucitol on the sphincter and to overcome biliary tract spasm. It was given initially in a dosage of one teaspoonful 3 times daily, ½ hour before meals. The patients were observed at least once weekly for from 2 weeks to 2 months and their progress recorded. During this time the dosage was adjusted according to their needs. The therapeutic results were tabulated as 0 (worse), 1 (no improvement), 2 (moderate improvement), and 3 (marked improvement), for each symptom and as an overall index of clinical progress.

TABLE II

CLINICAL RESPONSE

Symptom	0	1	2	3	Total
Fat intolerance	0	2	20	5	27
Nausea	1			4	5
Abdominal discomfort	0	2	18	10	30
Eructations	0	3	22	7	32
Pain	0	2	0	2	4
Constipation	0	5	15	12	32

RESULTS

The over all therapeutic results were: no increase in severity of symptoms in any of the patients, no improvement in 5 (13.1 per cent) moderate improvement in 18 (47.3 per cent), and marked improvement in 15 (39.4 per cent). Of the entire series, therefore, 33 (or 86.7 per cent) showed unequivocal clinical improvement of a substantial degree.

The over all results in each of the four biliary tract diseases studied are shown in Table I. The least improvement occurred in the 5 cases of cholelithiasis and the most in the 7 cases of delayed emptying of the gallbladder. Considerable improvement occurred in chronic cholecystitis without stones and the postcholecystectomy syndrome. It is worth noting that all cases of delayed emptying and postcholecystectomy syndrome reported good to excellent symptomatic improvement. In one case where the gallbladder did not visualize on the cholecystography immediately before therapy was started, a repeat cholecystogram at the end of IsoBilagol therapy showed good contraction and evacuation.

The effects on each of the symptoms studied is shown in Table II. The symptoms most greatly benefited were fat intolerance, abdominal discomfort, constipation, and nausea, although the only patient who suffered an untoward effect from the medication stated that her nausea had increased. Two patients reported marked improvement in right upper quadrant pain and 2 reported none at all. Of the latter, 1 had cholelithiasis and 1 had a nonvisualized gallbladder and a possible pancreatitis; of the former, 1 had cholelithiasis and 1 nonvisualization of the gallbladder.

Comment

In general it can be stated that a good graded clinical improvement occurred during therapy with IsoBilagol in many instances in patients who had not responded to other types of therapy. In many of the cases showing no improvement, the medication was discontinued by the patients after only 2 weeks of the projected 8, and it is probable that some improvement may have become manifest had the patients persisted in their therapy. On the other hand, in 16 patients (10 with chronic cholecystitis, 4 with postcholecystectomy syndrome, 1 with cholelithiasis, and (with delayed emptying), improvement was sufficient to allow a reduction of dosage after 1 to 3 weeks. In most cases clinical improvement continued for months after the medication had been discontinued altogether. Such results as were obtained in this preliminary study indicate that IsoBilagol is a very promising cholagogue and merits further and more definitive study.

Summary and Conclusions

1. A group of 38 patients with biliary tract disease were treated with a preparation containing d-glucitol and diisopromine (IsoBilagol).

2. Of these, none became worse; 13.1 per cent showed no improvement, 47.3 per cent moderate improvement, and 39.4 per cent marked improvement.

3. Significant statistical improvement occurred in those cases with chronic cholecystitis with no evidence of stones, delayed emptying of the gallbladder, and postcholecystectomy syndrome; equivocal statistical improvement occurred in the 5 cases of cholelithiasis.

4. Significant improvement was noted in fat tolerance, nausea, abdominal discomfort, eructations, and constipation; improvement of a lesser degree occurred in the cases with pain.

5. Except for one patient whose nausea increased, none of the patients showed any toxic effects.

6. IsoBilagol is a good, well-tolerated cholecystokinetic for the treatment of biliary tract disease and merits further study.

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CLONORCHIASIS
INFECTION CAUSED BY CLONORCHIS SINENSIS

Report of a Case

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Clonorchiasis, an infection caused by the infestation of the bile passages by *Clonorchis sinensis*, the oriental liver fluke, is acquired by eating raw or imperfectly cooked fresh-water fish which harbor encysted metacercariae. Incidence of the disease is rare in this country, as attested by the scarcity of reports of cases in the literature from America.

According to Craig and Faust[1], Belding[2], and others, the fluke was first discovered by McConnell in 1875, in the bile passages of a Chinese patient in Calcutta. It was named *Distoma sinense* by Cobbald in that same year. For the most part the disease is indigenous to the Far East including Japan, southern and central China, Formosa, Korea, and French Indo-China. It is also found in the Hawaiian Islands, but here it is ascribed to eating infected fish imported from endemic areas of the Far East.

Our purpose is to stress differential diagnosis in examining patients from endemic areas presenting hepatic, epigastric, and abdominal symptoms, and with a history of having eaten raw or insufficiently cooked fresh-water fish. Secondly, to emphasize that the presence of adult worms in the distal bile passages sets up considerable serious, sometimes rapidly developing pathology and histopathology, especially in the left lobe of the liver, and is often the cause of superimposed complicating diseases. We are reporting the case of a patient with clonorchiasis recently under our care, which demonstrates some of these salient points.

Snails are the first intermediate hosts of *Clonorchis sinensis*, second are certain species of fresh-water fishes, and man and other mammals are the definitive hosts in which the fluke may live for up to 20 years. The larvae hatch in the digestive tract of the snail, and pass through stages to miracidia to sporocysts, and then to rediae which produce cercariae. After their escape from the snail, and a free-swimming existence, the cercariae become attached to the

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fish, discard their tails, penetrate under the scales and form metacercariae, and encyst either in the skin or in the flesh. When the infected fish is eaten by man and other mammals, the metacercariae excyst in the duodenum, and migrate through the ampulla of Vater into the distal bile passages, where they mature into adult worms. The life cycle of *Clonorchis sinensis* is illustrated in Figure 1.

The adult worm is a flat, transparent, flabby organism, approximately 10 to 25 mm. in length, and 3 to 5 mm. in breadth. It usually lives in the smaller bile ducts, but may migrate, or be carried by the current of bile to the larger ducts, at times to the common duct. Hou Pao-Chang[3] reports 500 necropsy cases of *Clonorchis sinensis* in China. He states that at surgical exploration, or at autopsy, the common bile and hepatic ducts are often found filled with adult worms; in cases of heavy infestation the number in the common duct often

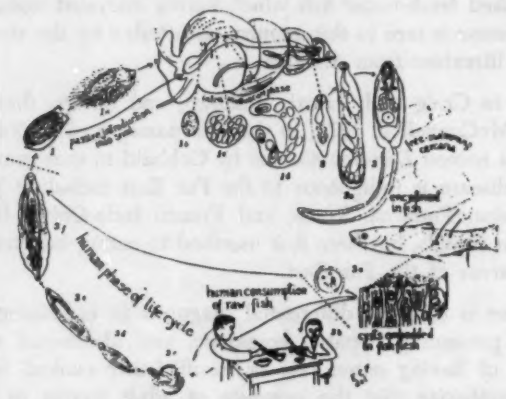

Fig. 1—Diagram of the life cycle of *Clonorchis sinensis*. 1, 1a-1d, First generation (i.e., egg ⟶ miracidium ⟶ sporocyst); 2, 2a, second (i.e., redia) generation; 3, 3a-3f, definitive generation (i.e., cercaria ⟶ encysted metacercaria ⟶ excysted young worms ⟶ adult worms). (After Faust)
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exceeding one hundred. The ova of *Clonorchis sinensis* are deposited in the larger bile passages, travel down from the biliary tract to the intestinal tract, and are evacuated in feces.

Primary lesions produced by the presence of this fluke are caused by its irritative action and toxic effects, particularly in long standing infections. They include a proliferative and inflammatory reaction in the biliary epithelium with its subsequent desquamation, a thickening of the walls of the ducts, crypt formation and development of new bile capillaries, periepithelial fibrosis, and periportal hyperplasia of connective tissue. According to Craig and Faust there is also development of tissue around "graves of eggs" infiltrated in the liver

parenchyma. After prolonged infection there is believed by many authors, to be a marked tendency to pressure necrosis, and periportal cirrhosis.

Other resulting pathology reported includes splenic hypertrophy, multiple abscess formation, cholangitis, cholangiohepatitis, intrahepatic calculi, and sometimes invasion of the pancreatic duct by *Clonorchis sinensis*. In addition, malignant alteration in the adenomatous tissue of the bile ducts, and hepatomas have been reported as serious consequences of the disease.

Hou Pao-Chang states that obstructive cholangitis is usually followed by biliary stasis, and that bile is toxic to tissues unprotected by epithelium. He

TABLE I

LIVER PROFILE

Dates	Icteric Index (Units)	Cephalin Flocculation (Units)	Thymol Turbidity (Units)	Total Proteins mg./100 c.c.	A/G Ratio	Bromsulfalein Test At 45 min.	Alkaline Phosphatase King Armstrong Units	Prothrombin Time	Activity (Seconds)	Control (Seconds)
2-21	9	2+	8.8	6.05	3.40 2.65	98.7	17.8	14.7	58.3	12.2
2-28						93.3	30	11.6	100	12.1
3-13	50	3+	10	6.25	2.95 3.30		34	15.8	43	11.0
3-20				4.45	2.65 1.80			16.1	52.9	12.6

points out that in clonorchiasis stagnant bile, as a rule, contains metabolic products of the live worms and disintegrated products of the dead ones. This favors bacterial infection, usually by *Escherichia coli*, leading to back flow of bile into the smaller branches of the bile ducts, and resulting in cellular infiltration of the portal spaces.

Otto[4] emphasizes catarrhal cholangitis as a serious pathologic change in clonorchiasis, caused by obstruction of the large bile passages by sticky masses of eggs, and tissue proliferation, Otto, Belding, and others, also attribute the cause of a systemic toxemia, including palpitation, tachycardia, vertigo, tetanic cramping, and sometimes a mental depression, to impairment of the detoxifying properties of the liver by the worms and their by-products.

In 1949, Koenigstein[5], studied clonorchiasis in representative patients of a group of 20,000 Jewish refugees to Shanghai, China, with a history of having eaten pickled fish. In early examinations of some of these patients he found an enlarged tender liver, slight icterus, splenomegaly, and eosinophilia. In examinations a few weeks later, he observed a chronic stage of the disease with symptoms of cholecystitis and hepatitis.

Clonorchiasis follows a chronic course and progresses with the duration of the infection. In some cases there are periods of remission. The disease increases in severity according to the number of worms, the average number, according to the literature, varying from 20 to 200. As many as 21,000, however, have been reported recovered from one patient.

Early symptoms include indigestion, epigastric distress not related to food, sometimes a marked leucocytosis, eosinophilia, and an anemia of varying degree. Night blindness has also been mentioned as a symptom. In more advanced infections, the symptoms may include edema, diarrhea, hepatomegaly, ascites, and cachexia. Icterus is not always present.

Diagnosis is made by discovery of the characteristic eggs of *Clonorchis sinensis* in the feces or duodenal contents. The eggs are markedly ovoidal, with a moderately thick, yellowish-brown shell. They have a convex cover which rests into a rimmed extension of the shell. At the thickened posterior end there is often a short comma-shaped protuberance. The eggs measure approximately 27 to 35 microns by 12 to 20 microns, the average about 29 by 16 microns.

Clonorchiasis, however, must be differentiated clinically from such pathologic conditions as idiopathic cirrhosis, echinococcosis, and severe beri-beri in patients newly arrived from endemic areas of the Far East, where the diet is largely limited to rice. Furthermore, with the assumption of some authors that the disease predisposes to malignancy, a case of long standing must be carefully examined to rule out a neoplasm.

Report of a Case

J. L., a 36-year old Chinese male, born in Hong Kong, China, had been living in this country for the past five years. He had been hospitalized on another service at Mercy Hospital in Chicago, from 28 January to 5 February 1957. During this time complete roentgenograms of the gastrointestinal tract were made which revealed no evidence of tumor or ulceration. The patient was referred to our surgical service at Mercy Hospital on 20 February 1957, for an exploratory operation. He complained of increasing abdominal distention, epigastric and right upper quadrant pain occasionally radiating to both flanks under the costal margin, not relieved by alkalis; postprandial upper abdominal fullness; nausea and occasional emesis; and a frequent urge to defecate, with the passage of several soft stools daily. He denied any excessive alcohol intake.

Past history disclosed that 20 years previously, in China, the patient had suffered abdominal pain and discomfort which was somewhat prolonged, but subsided spontaneously. He had been seen by a physician at that time, but stated that no diagnosis had been given him. The patient also stated that in the intervening period he had experienced mild occasional spells of abdominal discomfort and pain, epigastric fullness, and belching.

Physical examination revealed the abdomen markedly distended, umbilicus flat, and the abdominal skin shiny and tense. A very tender, round, epigastric mass was palpable. Liver dullness was percussed 2 to 3 fingers below the right costal margin. There was a fluid wave and shifting dullness. Further abdominal palpation was difficult because of the ascites. A one plus pitting edema of the lower extremities was present. A roentgenogram of the chest revealed pleural reaction and slight infiltration in the right base. An intravenous pyelogram showed an aplastic left kidney, and a hypertrophic right kidney.

We reviewed the gastrointestinal tract films taken in January 1957, and also made a second roentgenographic examination. This revealed only a mild cardiospasm; no evidence of ulcer or tumor. Proctoscopic and gastroscopic examinations were attempted, but were discontinued as the patient could not cooperate because of his weakened condition. A roentgenogram of the gallbladder, however, was accomplished; it filled normally and revealed no calculi or evidence of other pathology.

On 21 February, the day of admission, blood count showed 18,000 white blood cells, 3,670,000 red blood cells, and hemoglobin 12.4 gm. per 100 cc., 79.5 per cent. Differential count revealed 67 per cent polymorphonuclear leucocytes, 63 per cent segmented and 4 per cent nonsegmented neutrophils, 8 per cent eosinophils, 14 per cent lymphocytes, and 11 per cent monocytes. Results of a glucose tolerance test showed fasting 80 mg. per cent, one-half hour 127 mg. per cent, one hour 140 mg. per cent, two hours 167 mg. per cent, three hours 145 mg. per cent, and four hours 140 mg. per cent. Urine specimens were all negative for glucose and other findings. Blood urea nitrogen was 25 mg. per cent.

On 4 March, the day prior to a scheduled exploratory operation, the patient had two closely successive stools. These were examined and found positive for *Clonorchis sinensis*. This finding was first discovered by one of us (G.K.) resident on our medical service, confirmed by the pathologist at Mercy Hospital, and again by a parasitologist, Dr. Carroll L. Birch.

On 7 March the blood count showed 18,500 white blood cells. On 8 March a paracentesis was performed; 4,000 c.c. of straw-colored ascitic fluid was negative for ova, parasites, tumor cells, or tuberculosis. Specific gravity was 1.014.

A liver biopsy on 11 March revealed slight distortion of lobulation with marked lymphocytic infiltration of periportal spaces; no parasites.

By 12 March a blood count showed an increase in white blood cells to 20,850. On 13, 14 and 15 March stools and ascitic fluid were again negative for ova, parasites, tumor cells, or tuberculosis. On 16 March, however, stools were found positive for *Clonorchis sinensis*, and in addition *Heterophyes Heterophyes* were also discovered in the feces by the previously mentioned parasitologist, Dr. Carroll L. Birch.

On 18 March the white blood cells had increased to 29,250; chlorides were 96 mEq., potassium 5.52 mEq., and sodium 120.7 mEq., per liter, and CO_2 capacity was 27.0 Results of a stool test 20 March revealed 4 plus occult blood, but were negative for ova, parasites, tumor cells, or tuberculosis. Blood urea nitrogen was 60 mg. per cent, CO_2 capacity 39, and chlorides were 98 mEq., potassium 5.23 mEq., and sodium 122.7 mEq., per liter.

On 22 March blood count showed 25,150 white blood cells, 3,050,000 red blood cells, and hemoglobin 10.2 gm. per 100 c.c., 65.3 per cent. Differential count showed 85 per cent polymorphonuclear leucocytes, 84 per cent segmented and 1 per cent nonsegmented neutrophils, 9 per cent lymphocytes, 6 per cent monocytes, and the eosinophils had gradually decreased, until by this date they had reached a reading of one per cent, an atypical feature in this disease. Blood urea nitrogen, and the chlorides, potassium, and sodium remained unchanged. Calcium was 8.4 mg. per 100 ml., and phosphorus 5 mg. per 100 ml.

A paracentesis performed on 25 March revealed 6,000 c.c. of bile stained fluid negative for ova, parasites, tumor cells, or tuberculosis, and stool tests 26 and 27 March also showed negative findings. On 27 March the CO_2 capacity was 31, blood urea nitrogen 70 mg. per cent, and hematocrit was 22. Roentgenograms on 26 and 27 March showed a large amount of fluid in the right chest.

The patient died on 27 March from acute hepatic failure, a severe cholangitis, and electrolyte imbalance. We believe that other pathology was also involved in the cause of death, but our request for a postmortem was refused.

Comment

It was interesting to note that in addition to *Clonorchis sinensis*, *Heterophyes Heterophyes* were also discovered in the patient's stool. This fluke, which produces heterophyiasis, attaches itself to the wall of the small intestine and intestinal mucosa. Like *Clonorchis sinensis*, it is indigenous to the Far East. Craig and Faust point out that it is difficult to differentiate this species in the egg stage from those of other species of heterophyid eggs, and that they somewhat resemble those of *Clonorchis sinensis*. Likewise, it was interesting that eosinophilia, usually typical in clonorchiasis, was not prominent in our case.

As evidenced by the laboratory findings, particularly those of the last few days of life, our patient died in acidosis. We attributed this to his constant diarrhea, sometimes 15 to 20 stools a day, eventually associated with frank

hemorrhage from the bowel. Adequate intravenous blood replacement was made, and large amounts of intravenous fluids with sodium and chlorides were administered. In spite of this, it became an increasing problem to keep the electrolytes in proper balance. It was our feeling that the patient's elevated blood urea nitrogen and depressed CO_2 capacity, represented the end stage of irreversible hyponatremia.

The patient had been treated symptomatically, receiving abundant antibiotic therapy including tetracycline, chloramphenicol, penicillin, and streptomycin, to affect the cholangitis. In addition we administered Vitamin K, 50 mg. daily, to improve the prothrombin time. The treatment of *Clonorchis sinensis* infection *per se*, however, is not very satisfactory. Gentian violet, orally or by intraduodenal instillation, is sometimes effective in early stages; in late or in chronic cases it will often diminish the number of worms present. Tartar emetic (potassium antimony tartrate), tetrachloroethylene which is used in hookworm infection, and nonsurgical biliary drainage have likewise been employed. Chloroquine diphosphate (Aralen diphosphate®), has been found useful in the therapy of clonorchiasis when given over a period of 20 days.

A course of Chloroquine diphosphate (Aralen diphosphate®) was started for our patient, and continued for 10 days (1 gm. daily). He could not, however, tolerate the drug and nausea persisted. We then used gentian violet for 5 days, but as nausea continued, all medication of this type was discontinued. Therefore, an estimate of the results that could possibly have been obtained from these two drugs in combating the severe infestation, was not available to us. We feel, however, that the negative stool findings in the latter period, represent some response to therapy.

From the patient's condition on admittance, his hospital course, and the assumed diagnosis at death, without surgical exploration or benefit of autopsy, it was evident that he had undoubtedly become infected with *Clonorchis sinensis* many years prior to his last illness. Although unfortunately, the date of inception of the infection could not be documented, several factors would potentially designate the case as one of long standing infection, with considerable complicating pathology: 1. the patient's past history of abdominal pain and distress 20 years previously, 2. intermittent attacks since that time, 3. tendency of the disease toward progression, remission, and recurrence, and 4. the fact that the infection can persist in man for up to 20 years, even after leaving an endemic area.

The assumption of long standing infection was further substantiated by the fact that from the patient's course, it was obvious that a severe cholangitis had set in. Furthermore, recognizing the possibility of a malignancy in advanced cases of clonorchiasis, and although we had a liver biopsy which revealed no evidence of carcinoma, we feel, nevertheless, that a hepatoma may have been associated with the parasitic infection. Belding cites the opinion of Hoeppli[6] that

clonorchiasis predisposes to malignancy. In this regard, Labby[7] states that hepatomas of the liver constitute 14.1 per cent of all carcinomas in the Orient. This is a considerable increase over that seen in this country or in Europe. Furthermore, Labby points out that since there appears to be a higher incidence of primary malignancies in Orientals than seen elsewhere throughout the world, it might be suggested that parasitic infestation may account for this higher figure.

The relationship between clonorchiasis and cirrhosis has also been discussed in the literature, with some varying opinions. For example, Belding states that with continuous reinfection by *Clonorchis sinensis*, a serious cirrhosis of the Laennec type may ensue, with extensive destruction of the liver parenchyma. As stated previously, some authors point out that after many years of infection, there is a marked tendency to periportal cirrhosis. Cecil[8] states that 2 to 3 per cent of all autopsies in the United States show a Laennec cirrhosis, and 2 to 10 per cent in the Orient.

On the other hand, in the opinion of Hou Pao-Chang, there is no anatomic evidence to support an exact relationship between clonorchiasis and multilobular cirrhosis; that none of the livers examined by him showed evidence of diffuse cirrhosis of this type; initiated by *Clonorchis sinensis*. He states that: "Statistically in 500 cases of clonorchiasis there were 45 cases of multilobular cirrhosis (9 per cent). In a group of 13 cases of biliary cirrhosis 5 (38.4 per cent) were associated with clonorchiasis. This figure may seem high," he states, "but since 46 per cent of livers in routine postmortems were infested with *Clonorchis sinensis*, this figure is only to be expected." On the basis of Hou Pao-Chang's statistics, it would appear that hepatic cirrhosis is not a common concomitant of clonorchiasis.

Orientals living in the Western Hemisphere have also been parasitized by *Clonorchis sinensis*. From the scarcity of reports of the infection in this country, however, evidence is lacking that it has become established in this hemisphere. Nevertheless, this progressive disease has serious implications, especially when diagnosis and treatment may have been long delayed. It occurs to us, therefore, that clonorchiasis should be considered as a possibility, not alone in orientals, but in soldiers of the Western Hemisphere stationed in the Far East, those recently discharged from service in Korea, China, Japan, and other Far East countries, and native Americans as well, newly arrived from endemic areas.

Summary

A case of clonorchiasis, an infestation of the bile ducts by the parasite *Clonorchis sinensis* is presented. This oriental liver fluke causes dilatation of the biliary ducts and intrahepatic portal veins. The disease is indigenous to the Far East and its occurrence is rare in the Western Hemisphere.

Importance of differential diagnosis is emphasized in the presence of hepatic, epigastric, and abdominal symptoms in patients from endemic areas.

Symptoms of the disease, and the pathologic changes initiated by this fluke, are described. It is explained that these are usually caused by obstruction of the main hepatic or common bile duct, and aggravated, in some cases, by bacterial infection.

Opinions on the relationship of clonorchiasis to cirrhosis and malignancy are discussed.

Results of laboratory studies in the authors' case, particularly liver function tests, are outlined.

Treatment of the disease is discussed. Various recommended therapeutic drugs are listed, and it is pointed out that *Clonorchis sinensis* infection, in early stages, may be benefited by drug therapy.

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MANAGEMENT OF THE OBESE PATIENT

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In the past year I have had the opportunity to treat 40 adults who came in requesting weight reduction. Medical authorities, insurance companies and health agencies are now convinced that excess weight is a physical, social and psychological hazard[6]. It is estimated that one in every five adults over the age of 30 is overweight[5]. One needs only to study insurance statistics to realize the great increase in morbidity and mortality in the overweight[6]. In adults who are overweight, there is a 51 per cent greater incidence of cardiac disease; 65 per cent increase in vascular disease; a 250 per cent increase in diabetes and a 67 per cent increase in cirrhosis of the liver[1,6].

The patients who came in and asked to be aided in reducing fell into the following categories:

1. Simple overweight.

2. Overweight with symptoms of fatigue, shortness of breath, and tiredness. In these cases, no pathology was found.

3. Overweight with symptoms plus definite pathology. The most common findings were gallstones, coronary artery disease, hypertension and arthritis.

These patients had a complete history, physical examination and laboratory work done. The history of the patient's dietary habits was recorded. The patient's proper weight was determined and each was classified as to the cause of his or her obesity. The reason for reduction was explained to the patient. Blood-pressure readings were particularly watched and individual and collective weight recordings were stressed. In each case we had to determine how fast these patients were to be reduced, what drugs if any were to be used. Precise written instructions were given to each patient. A card with a graph was made out for each patient and put on the bulletin board to be compared with other patients. In some cases, weekly, and in others monthly visits were requested. A follow-up after the patient ceased to take drugs was made.

It was found that all patients had the following factors concerning their overweight.

1. The most common factor was eating too much at meals. This was particularly found to be true of the evening meal.

2. The second most common factor was the snack habit. This included eating at parties and while watching television.

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3. The least common factor was eating foods that contain too many calories.

4. In some patients a combination of all three was found.

When a patient was found to be overweight, specific written instructions were given, depending on the category. For example, Class A were given the following instructions:

"You are overweight because you eat too much at meals, particularly the evening meal. You are not to satisfy your appetite completely. You are to have smaller portions. You are not to have any desserts. Get away from the table after the main course. Do not indulge in sauces; trim off fats; never take a second helping. Take one Ambar Extentab* before breakfast. Weigh yourself every four days; mark it down on the chart provided. You must lose one pound every four days. If you do not lose that amount, you must cut down on your meals. Bring your chart and report to the doctor weekly."

Instructions given to Class B followed a similar line. "You are overweight due to faulty snack habits. Do not nibble between meals. Remember eat only at meal time. No food must be taken at night."

Instructions to those belonging to Class C were as follows. "You are overweight because the food that you eat contains too many calories. You are not to have any butter, salad dressings, creamed sauces, fried foods, gravy, fat soups, fat meats, alcoholic drinks, pies, pastries, cookies, candies, chocolates, soft drinks, cream, sugar, starchy foods, no desserts, limit bread to a minimum."

In conjunction with the simplified classification and accordingly simplified diet instructions, it was found that nearly all patients needed a "crutch". This crutch was necessary to overcome the abnormal desire to eat frequently; to curb an abnormal appetite, and to relax those that were under tension. It was found that Ambar Extentab was an ideal "crutch" because of its "mood and appetite control" and because of its extended action. This tablet contains: Methamphetamine hydrochloride, 10.0 mg.; phenobarbital (1 gr.), 64.8 mg. This tablet has an extended action of uniform effect for 12 hours. Thus one tablet before breakfast was sufficient for most of the waking day. For the above reasons, Ambar Extentab was found entirely satisfactory and practical in this study.

Forty cases constituted the Ambar-diet study in this series of obesity. Five cases had to be discarded because of their failure to follow the routine and their failure to return. The 35 remaining cases were followed very closely. The following four cases are chosen and details are given.

Case 1:—Mrs. A. A., age 38, female, weight in April 1957, 210 pounds; ideal weight, 140 pounds; original blood pressure, 150/100; last blood pressure,

*Ambar Extentabs were kindly supplied to us by A. H. Robins Company of Richmond, Va., U. S. A.

130/80. This patient, in my opinion was 70 pounds overweight, and definitely has gallbladder disease. It was decided to reduce her weight because of her mild hypertension and because she was a poor risk for surgery.

It was decided that her overweight might be corrected if she ate less between meals and at meal time. The patient was put on a routine of one Ambar Extentab in the morning and one vitamin pill daily. She was asked to lose five pounds a month. Instructions were given to her as to her diet and routine.

The results are shown in Figure 1. She was taken off Ambar when she reached 165 ponds. Her latest weight was recorded in February 1958 at 160 pounds.

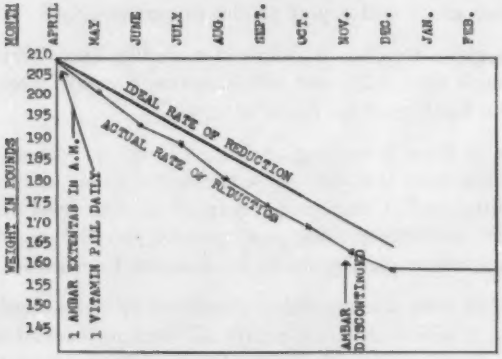

Fig. 1—Case 1, Mrs. A. A.

Patient has learned to control her food intake. She has lost 45 pounds. She feels well. Her latest blood pressure is 130/80. It is hoped that this patient will be able to have her gallbladder removed in the Spring of 1958.

In my opinion, this case represents an ideal result. It has helped her gallbladder symptoms. Her blood pressure has now been reduced and generally she feels well.

Case 2:—Mrs. M. S., age 52, female, weight in February 1957, 165 pounds; ideal weight, 145 pounds; original blood pressure, 140/80; last blood pressure, 110/70. This patient complained of tiredness, weakness. She has gained 20 pounds in the last two years. She had many complaints with reference to her gastrointestinal system, and her gallbladder.

Physical examination was essentially negative. She had a poorly functioning gallbladder and a spastic colon. Her blood pressure was 140/80. Her E.C.G. was normal. Urine was negative, and no other abnormalities were found.

We decided to reduce her weight because of her 20 pounds overweight and poorly functioning gallbladder.

She was put on a regime to lose five pounds a month. It was decided that her increase in weight was a result of eating between meals. This faulty snack habit and her meals were regulated. She was put on one Ambar Extentab in the morning and one vitamin pill daily. She was to weigh herself every week and to try and lose two pounds a week. She was to return to the office monthly. She had no ill effects during the course of her reduction. Figure 2 shows her progress.

She was taken off Ambar Extentab when she reached 135 pounds. Since then her weight has been maintained between 135 and 140 pounds. At the last examination in December 1957, her weight was 136 pounds.

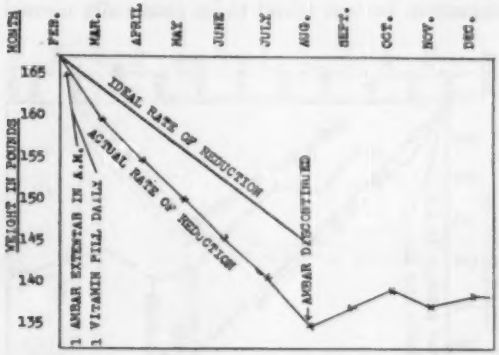

Fig. 2—Case 2, Mrs. M. S.

As a result of her weight loss, she feels better mentally and physically. The patient was advised as to the importance of staying on the routine and to get in touch with me as problems arise.

In my opinion, this is a typical excellent result.

Case 3:—Mr. J. M., age 54, male, weight in October 1956, 209 pounds; ideal weight, 170 pounds; original blood pressure, 120/80; last blood pressure, 110/80. This patient was put on reduction because he was beginning to have some shortness of breath; he was much overweight and he has been increasing in weight lately.

Physical examination showed an obese, short individual with no definite abnormalities found. He was put on a regime to lose five pounds a month. His increase in weight was determined to be due to large meals and food between meals. He was instructed regarding eating smaller meals. He was forbidden to

eat between meals. He was given one Ambar Extentab to be taken in the morning together with a vitamin pill. He was to weigh himself weekly. He returned to the office for follow-up and a chart (Fig. 3) was kept of his weight.

There were no ill effects from taking the Ambar Extentabs or from dieting. Results of his weight loss is noted above. He was taken off Ambar in May 1956. His last weight in October 1957 was 189 pounds.

The results are good substantial gradual reduction in weight. No side-effects.

Case 4:—Mr. S. W., age 35, male, weight in December 1956, 185 pounds; ideal weight, 170 pounds; original blood pressure, 150/90; last blood pressure, 140/80. This man was found to be at least 15 pounds overweight. He came in for an insurance policy and this was refused him because of his excess of weight. On physical examination, he was found to be essentially normal except that his

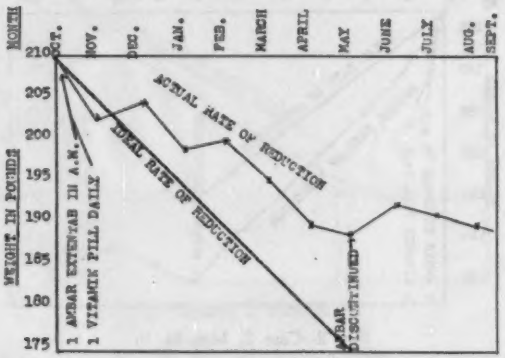

Fig. 3—Case 3, Mr. J. M.

blood pressure on repeated examination was found to be 150/90. His E.C.G. was normal.

On questioning him, it was found that he ate too much at meal times and that he ate between meals, particularly at night while watching television. He was put on a regime to lose five pounds a month. He was instructed to take one Ambar Extentab in the morning and one vitamin pill daily. It was explained to him that an essential part of the routine was to eat smaller meals and not to eat between meals. He has stated that the taking of Ambar sort of spiked his appetite and that he did not have to eat between meals. As indicated in Figure 4, the patient reached 170 pounds in March 1957.

At this point, he was taken off the Ambar. His latest weight in September 1957 was 170 pounds. He is obviously maintaining his reduced weight without

Ambar Extentabs. His latest blood pressure is 130/80. He feels well and he has no side-effects.

In the 35 patients studied, it was thought that the most important factors in maintaining their obesity were as follows:

1. Eating excessively between meals—12 patients.

2. Eating foods too high in caloric value—4 patients.

3. Eating too much at meal time—8 patients.

4. A combination of eating between meals and too much at meals—11 patients.

Most patients found that cutting out foods between meals, particularly in the evening was difficult to follow. These patients had a peculiar nervousness

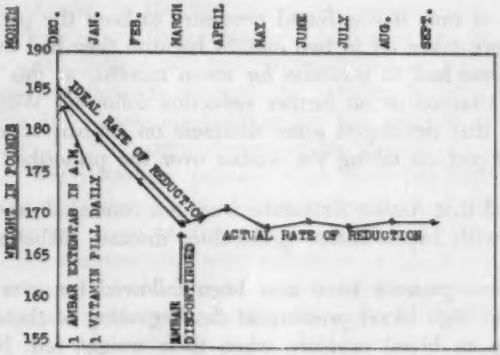

Fig. 4—Case 4, Mr. S. W.

and a psychological craving for food. This was reduced by taking Ambar Extentabs. This medication gave them a more buoyant and contented sensation. The patients who ate too much at meals were the easiest to put on regime, the desserts were taken away, less food was offered in the evening meal. Nearly all the patients were able to follow the simple instructions given. The Ambar Extentabs were well tolerated and the convenience of taking one tablet daily was appreciated. In the entire series, only two women complained of slight dizziness.

The weight loss per month, on an average, varied from three and a half to four and a half pounds. One patient lost 15 pounds in the first month and then slowed down to five pounds a month. Two patients lost ten pounds per month, then slowed down to four pounds a month. One patient did not lose any weight the first month and began to lose on an average of two pounds in the following months.

The most important reason for reducing the weight of some of these patients was the fact that they were overweight and had some associated symptoms and diseases. The most frequent of these were hypertension, arthritis, diabetes and gallbladder disease. Accordingly it was important to reduce these patients to near normal and keep them there after the medication was stopped. I followed 19 patients out of the 35 after they had ceased to take Ambar in any form. Some of these patients have been followed for six months or more. Only two patients out of the 19 which were followed after the Ambar was stopped still continued to lose more weight. Two patients began to increase their weight. Fifteen patients were able to maintain their weight loss. As can be seen, the majority of the patients had learned the reason for their overweight, controlled their diet, snack habits, large meals and were able to maintain their weight loss without drugs. This is a very significant part of this study. It proves that weight can be controlled by proper eating habits.

The length of time it was found necessary to keep the patients on Ambar varied. Some were taken off in two months because they had attained the goal set for them. Some had to continue for seven months; at this time either the goal had been attained or no further reduction followed. With the exception of two patients that developed some dizziness on Ambar, no clinical signs or symptoms developed on taking the Ambar over the prescribed period.

It was found that Ambar Extentabs were not contraindicated when obesity exists together with hypertension, gallbladder disease, diabetes or arthritis.

Some of these patients have now been followed for over a year. Those patients who had high blood pressure at the beginning of their reduction routine had a drop in blood pressure when their weight fell. None of the 35 patients followed in this series developed a rise in blood pressure. None of the patients had gallstone attacks; this includes the two patients who had proven gallstones before reduction was undertaken. The above beneficial side-effects suggest that in causing these patients to lose their excess of adipose tissue, we lowered their blood cholesterol and this benefited the patient as a whole and particularly coronary artery disease, hypertension and gallbladder disease.

The following are the general and particular conclusions that were observed in the course of this study. For the average weight control program, monthly observations were necessary. This acts as a psychological boost; it affords the physician an opportunity to evaluate progress and observe the results of his regime and medication. It also can be used as a competitive system to have the patients reduce in a group.

Anoretic and sedative medication, plus diet, plus encouragement forms the essence of the program. The regime suggested was simple and practical. It, in no way harmed or weakened the individual. It gradually reduced the

patient to the proper weight. The drugs given were well tolerated and harmless.

In thinking of outlining the regime, one must remember that most of these patients are not ill. They will not follow a complicated, exacting regime for any length of time. They refuse to walk around with diet sheets or caloric charts. If the regime is to be successful, it must be simple; it must fit the individual. If there is a nervous element or an element of terrific hunger, one must give the patient an anoretic and sedative medication.

In the above study, Ambar Extentabs were used for the anoretic and sedative effect. When the diet was etxensively reduced, a vitamin tablet was given daily.

There are little tricks to encourage the patient and make them stick to their prescribed regime. In the first place, the patient must want to reduce; the doctor must believe and be enthusiastic in his regime. A chart and graph is made out for each patient and put on the bulletin board. Frequent weighings and plotting, competition amongst the patients being reduced, adds zest and desire to succeed. Encouragement is the key note to success. Explain to the patient the long term program required for proper reduction. Explain the ups and downs and stationary periods. One must continue to follow these patients and keep reminding them of their rewards; remind them of their loss of ugly flesh; remind them of their improvement in looks and well being. Women are usually enthused by being reminded of smaller size clothing. It is also important to see that the weight loss be continued or remain stationary after the anoretic and sedative drug has been discontinued.

Summary and Conclusion

1. Forty adult patients were accepted for weight reduction. Five of these dropped out because of their inability to follow regime.

2. The results of the 35 others are given. Four histories are described in detail and graphs of the patients are presented.

3. Some of the obese patients were found to be suffering from other diseases with or without symptoms.

4. It was found necessary to classify the obese patients and outline treatment which varied in each classification.

5. All patients were given one Ambar Extentab in the morning. This helped them follow their regime, and it lessened their craving for food. It gave them a feeling of well being and controlled their psychological symptoms.

6. The results of weight reduction was followed in 19 patients after medication was discontinued. Here it was found that the weight was kept at the desired level after the drug was stopped.

7. Some beneficial side-effects were noted while patients were being reduced in weight. Particularly was this true of patients with cholecystitis, angina pectoris and hypertension.

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HEMORRHAGIC LEIOMYOMA OF SMALL INTESTINE SIMULATING GIANT OVARIAN CYST

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The small and large intestine not only present lesions which produce difficulties in their clinical differentiation from pelvic tumors, but also when unexpectedly encountered at operation, confront the gynecologist with the problem of challenging surgical technics. To avoid this situation, proctosigmoidoscopic examination and barium enemas as well as a study of the gastrointestinal tract with the motor meal have long been employed to help in the differential diagnosis. On the right side of the pelvis, appendiceal abscess and agglutinated bowel resulting from regional ileitis, present even more challenging aspects in their differential diagnosis. Personal unexpected encounter with appendiceal mucocele, liposarcoma of the omentum, lymphosarcoma of mesentery and small bowel have proved embarrassing. Most recently, a giant sized hemorrhagic leiomyoma of small intestine was clinically interpreted as a huge ovarian cyst. The case history of this uncommon lesion of small intestine is presented below. The complication of hemorrhage into the substance of the tumor accounted for its liquefaction and caused the error in diagnostic interpretation.

Report of Case

P. Z., female, age 70, was first seen by one of the authors (J. T.) on 30 December 1953. She complained of abdominal pains on and off of six months' duration. The pains were not sharp and had no relation to either food intake or bowel movements. Her appetite was poor and she had lost about 15 lbs. during that time. Her bowels moved daily and there was no history of rectal bleeding or melena. The patient claimed that for years she had suffered from heartburn and bloated feeling after meals.

Past history was negative except for chronic asthmatic bronchitis. Family history was irrelevant.

Physical examination revealed a blood pressure of 160/100 and weight of 153 lbs. There were sonorous rales over the entire chest anteriorly as well as posteriorly. The abdomen was enlarged. There was a palpable semisolid, nontender mass extending from the xiphoid process to the pelvis and laterally into

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each flank. Fluoroscopic examination of the chest was negative. Urine analysis was negative.

A provisional diagnosis of malignancy of the gastrointestinal tract was entertained and a gynecological condition ruled out.

Fluoroscopic and radiographic studies of the gastrointestinal tract done on 6 and 7 January 1954, revealed the following: The stomach was displaced upward. The small intestines as well as the colon were displaced upward and to the left. There were numerous diverticula in the descending colon and sigmoid. The above-described displacement of the gastrointestinal tract appeared to be caused by an extrinsic mass. There were no intrinsic defects (Figs. 1, 2 and 3).

A gynecological consultation was then ordered. The examination was done by one of the authors (S. A. W.). A diagnosis of giant ovarian cystoma of the

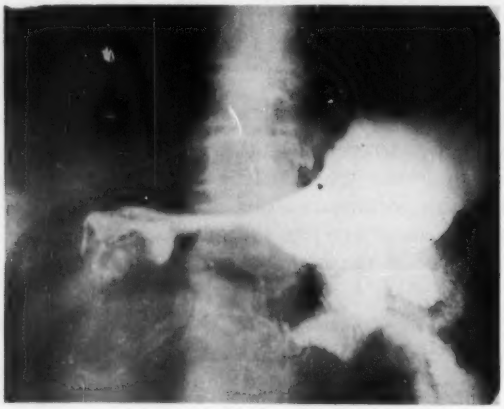

Fig. 1–Showing the stomach displaced upward.

pseudomucinous type was made and the patient was admitted to the Jewish Hospital of Brooklyn on 10 January 1954.

Operation was performed on 15 January 1954. A long, right paramedian incision reached from the symphysis to a level about 6 cm. above the umbilicus. The total length was about 17 cm. The abdominal cavity was found occupied by a huge, semicystic mass, reaching from the pelvis to the costal arches and reaching deeply into the flanks on each side. The lower pole of the tumor was fused with the anterior parietal and vesical peritoneum. Towards the left it was incorporated into the mesosigmoid. On the right it was relatively free. In front, however, it was firmly bound to the anterior abdominal wall by numerous, firmly organized adhesions. The superior pole was fused with, and incorporated into a segment of small bowel. The uterus and adonexae were not visible on inspection. The expected thin, translucent membrane of

grey white color, usually encountered with giant ovarian cysts was not present. Because of the huge size and fixity of the supposed cyst, the liquid contents were tapped by trocar and cannula resulting in the escape of 1,500 c.c. of hemorrhagic fluid. By sharp and blunt dissection, the lower pole was liberated from adjacent peritoneum and the mesosigmoid. Elevation of this segment of the tumor then revealed the normal underlying senile uterus and ovaries. Further mobilization followed transection of the anterior adhesions. The upper pole, however, was so intimately fused and incorporated into the intestinal wall that a segment of small intestine 10 cm. in length was resected *en masse* with the upper pole of the tumor. End-to-end anastomosis of the small intestine was

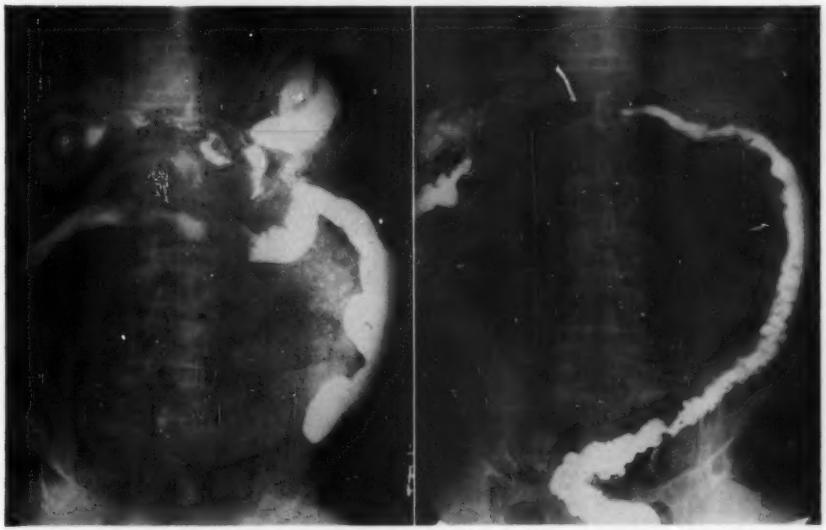

Fig. 2 Fig. 3

Fig. 2—Showing the small intestines displaced upward and to the left.
Fig. 3—Showing the colon displaced upward and to the left with diverticula in the descending colon and sigmoid.

then performed. The abdomen was closed in layers and a Levin tube inserted into the stomach. The postoperative course was without incident. The tube was removed on the 6th postoperative day. The wound healed *per primam* and the patient left the hospital on the 15th day following surgery. Follow-up examinations to date revealed the patient to be free from symptoms, weight fully regained, and physical examination free from abnormalities in the abdomen and pelvis.

Fluoroscopic and radiographic studies of the gastrointestinal tract done on 29 and 30 March 1954, were negative for pathology (Figs. 4, 5 and 6). Chole-

cystographic examination performed on 6 April 1954 disclosed a good functioning gallbladder with numerous small calcific shadows suggestive of lithiasis. This finding of cholelithiasis would probably account for the heartburns and bloated feeling after meals, mentioned in the patient's history.

The pathological report is as follows: The gross specimen (54-270) consists of a partially collapsed, cystic structure 24 x 12 x 15 cm. Fibrofatty tags are irregularly attached. Beneath the capsule and projecting into the central cavity are numerous closely set, small, round or oval, bluish mounds which on section consist of soft, pink, blue-grey tissue with a fibrillar texture. The lower pole which is lobular measures 8 x 5 x 3 cm. Centrally, it too presents a hemorrhagic

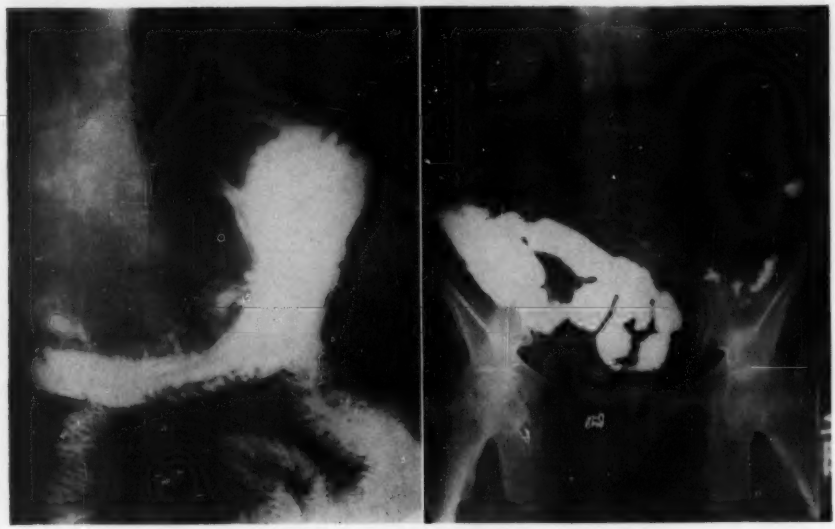

Fig. 4 Fig. 5

Fig. 4—X-ray revealing a normal stomach.
Fig. 5—Showing normal distribution of the small intestines.

cavity lined by yellow, soft, grey-pink tissue, which is fibrillary in pattern. Like the main cavity proper this small space is clothed with rough, brownish crumbling debris comprised of blood and disintegrating tumor. The segment of bowel attached to the upper pole measures 7 cm. in length. On its mesenteric border there is a segment of tumor measuring 4 x 3.5 x 2 cm. in size. Incision through the lumen reveals compression of the intestinal mucosa with a resulting ulceration 1.5 x 1 cm. in diameter. The muscle coats at the site of the tumor are largely disorganized. The serosa, too, is grey-white and hemorrhagic.

Microscopically, multiple sections prepared from varying areas through the tumor present a uniform histological pattern. The tumor is comprised of bands

of spindle cells coursing in parallel sheets, often interdecussating and even producing interlacing whorls (Fig. 7). The cell unit is large, spindle-shaped with abundant eosinophilic fibrillary cytoplasm. The nuclei are long, slender and present rounded ends. The nucleoli are not prominent, and the chromatin granules are uniformly distributed. Mitotic figures are only very occasionally encountered. By the Van Gieson stain, the cells are positively differentiated as those of smooth muscle type. In occasional areas the cells are shorter and plump. The nucleus assumes an ovoid pattern. There is, however, no variation in nuclear size, and shape. The occasional ones present increased

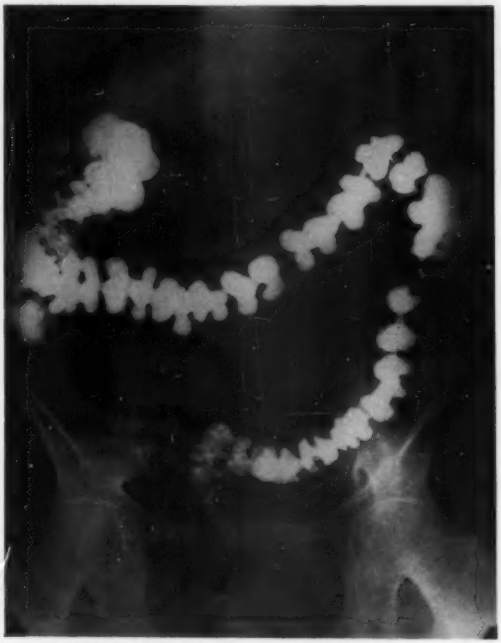

Fig. 6–Showing a normal colon.

staining intensity. Blocks from the small intestine reveal the lumen to be compressed by the tumor. The overlying mucosa is necrotic and the muscle layers are disassociated by growing tumor. The interior of the large and small cysts is closed with fibrin and necrotic debris, the underlying muscle containing large numbers of lymphocytes and numerous hemosiderin laden phagocytes.

Diagnosis:–Cellular leiomyoma of the small bowel; hemorrhage and secondary liquefaction necrosis.

Leiomyoma of the intestine warrants a brief description of its pertinent clinical and pathological manifestations.

Comment

In 1937 Smith[1] reviewed the literature and found that myomas occur more often in the small intestines than in any other portion of the alimentary tract with the exception of the stomach. He divided the myomas into intraluminal and extraluminal types because of certain clinical differences. The small intraluminal type is more likely to cause intussusception and intestinal obstruction but does not give rise to metastasis. The extraluminal myomas, however, growing into the peritoneum usually resulted fatally as a result of hemorrhage, perforation or metastasis. Some patients were saved by operation. According to Klepp and Crawford[2], the rate of growth of myomas is slow and the recurrences are often late. Horsley and Means[3], in 1955, collected 110 cases from the literature and added two of their own. Where the site of the tumor was specified, the lesion was distributed as follows: 38 tumors were found in the jejunum, 33 in the duodenum and 24 in the ileal segment of small bowel. Starr and

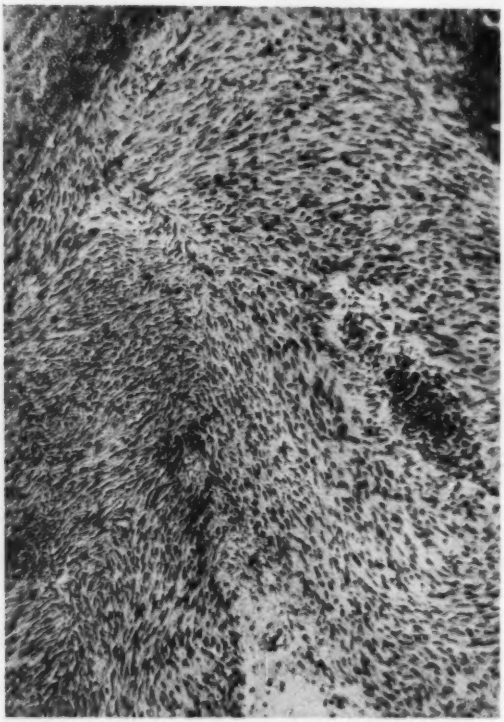

Fig. 7—The tumor consists of bands of spindle cells coursing in parallel sheets, which also often interdecussate.

Dockerty[4], in 1955, collected 230 cases from the literature and reviewed 76 benign and malignant leiomyomas encountered in their clinic. Raszkowski and Kent[5], added three jejunal leiomosarcomata among 65,638 surgical admissions to the Kings County Hospital between the years 1940 and 1951. J. S. Barb[6], reported a jejunal tumor which simulated a bleeding duodenal ulcer.

Both the benign and malignant muscle tumors of the small intestine have their origin in the muscular coat. As with uterine fibromyomata, the growth may be contained within the bowel wall and thus remain intramural. Those with growth direction towards bowel lumen are designated as endocentric, while those growing towards the serosa, are classified as exocentric lesions. According to Starr and Dockerty[4], the exocentric tumor is the most frequent form.

Leiomyoma and leiomyosarcoma of the intestine like similar muscle tumors elsewhere, are firm, tan or grey-white, nodular and fairly well encapsulated. The cut surface reveals grey tan whorls or interdecussating fusiculi. The surface vessels are always prominent. Multiple lesions may be encountered. The benign tumors are generally firmer in consistency and of smaller size. Complications are not uncommon. As in uterine fibromyomata, hemorrhage, liquefaction necrosis and occasional calcific degeneration occurs. Pressure on the overlying mucosa may lead to necrosis, with secondary infection of the tumor and fistula formation. In leiomyomas massive hemorrhage is a rather frequent occurrence and it may prove fatal. After a leiomyoma achieves a certain size it may undergo central hemorrhagic necrosis, becoming honeycombed with blood-filled cavities. These cavities may merge, leaving only a tumor shell, which finally perforates into the intestine, causing a gross hemorrhage. The malignant tumors are larger in size and generally softer in consistency. Spread is generally by local implantation. In tumors of the duodenum and jejunum, however, metastasis to the liver is not uncommon.

Microscopically, intestinal leiomyomata often present a challenge in the differential diagnosis of benign cellular forms, from early malignant types. As a rule, the tumor is composed of spindle-shaped cells with a fibrillary cytoplasm and a plump nucleus. The cells course in broad parallel sheets, intertwine or produce multiple whorls. In malignant forms, the cell differentiation becomes abnormal, the cells assuming a shorter, rounder form and the nuclei varying slightly in size, shape and staining capacity. The Van Gieson and phosphotungstic acid stains help to establish the identity of the muscle cell. In malignant forms palisading of the nuclei as seen in uterine myosarcoma is not uncommon. Where frank malignant aberration has occurred, pleomorphism with varying sized cells and irregular, hyperchromatic nuclei appear. Giant cells are numerous. The presence of mitotic figures holds a unique position in the grading of the malignancy established by Starr and Dockerty[4]. Thus, tumors of Grade I malignancy show one mitotic figure in one to ten high power fields; those of Grade II contain two to five mitotic figures per high power field. In neoplasms

classified as Grade III, more than five mitotic figures appear within each high power field. In Grade IV tumors, pleomorphism, cellularity, nuclear aberration and loss of polarity are dominant. In the Mayo Clinic series, 35 tumors were diagnosed as benign leiomyomata. In 31 cases, however, malignancy was established by the criteria described above.

The symptoms of leiomyoma and leiomyosarcoma are not distinctive. They resemble those produced by other lesions of small bowel. Starr and Dockerty[4], indicate that the lesion is more frequent in men than in women with a ratio of 2:1. The age of predilection of the disease ranges from 40 to 59 years. Pain is the most frequent complaint, but is of varying nature and intensity. Endocentric tumors are the cause of crampy pain resulting from ulceration, partial intussusception or altered peristaltic function. Exocentric growths, however, produce pain only by traction, rotation or impeded circulation. Intestinal bleeding or melena was noted in 5 of the 230 cases reviewed by Starr and Dockerty[4]; while Horsley and Means[3], encountered this in two-thirds of their series. It may be presumed that frequent bouts of melena are the result of ulcerated mucosa; while widely separated episodes probably result from mechanical factors of rotation or venous congestion.

The presence of an abdominal mass was noted in over half of the cases reviewed by Horsley and Means[3]. Small tumors, however, frequently escape detection because of their mobility and their location in the upper segment of the bowel. In the absence of a palpable tumor mass unexplained intestinal bleeding may be baffling and the cause is uncommonly ascribed to a gastric or duodenal ulcer. Complications of intussusception and intraperitoneal rupture with peritonitis may be the first indication of the presence of intestinal leiomyoma or leiomyosarcoma.

X-ray studies though unrevealing in our own personal case, are of great diagnostic value. This modality was useful in the diagnosis of 23 of 38 cases of Starr and Dockerty[4]. In the three jejunal cases of Raszkowski and Kent[5], one showed ulceration of the mucosa, one obliteration of the mucosal folds, while the third case showed an indefinable, extrinsic pressure effect. The deformity caused by the tumor is sharply defined at each end of the lesion.

The treatment of leiomyoma and leiomyosarcoma is surgical. Excision is the treatment of choice. Since the lymph nodes are not involved extensive dissection is not indicated. As reported by Starr and Dockerty[4], even with advanced peritoneal involvement, simple resection may produce arrest of the progress of the disease. In one of their patients metastases were accidentally discovered 13 years following resection, during the course of a subsequent hernial repair. The hernial sac and the peritoneum were found studded with metastatic deposits and these were irremovable. The patient, however, is reported to be in good condition 13 years following primary procedure and 6 years following the herniorrhaphy.

The prognosis of the benign lesions is good although in two of the cases reported by Starr and Dockerty[4], later recurrence indicated biological growth. Sarcomatous change in an unexamined segment of the tumor is the likely explanation. Of 15 frankly anaplastic tumors in their series 12 patients died. Long periods of remission, however, followed palliative removal of the primary growth.

Summary

A case of exocentric leiomyoma of the small intestine is reported. The presence of hemorrhage and liquefaction necrosis resulted in cystic changes. Erroneously this was attributed to the presence of an ovarian tumor. The symptoms, findings and clinical course of leiomyoma and leiomyosarcoma are reviewed. The problem in differential pathological diagnosis of benign and malignant leiomyomata is stressed.

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MESENTERIC THROMBOSIS ASSOCIATED WITH MIGRATORY POLYPHLEBITIS

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More than a decade ago, I reported my first personal experiences with mesenteric thrombosis. At that time I presented the studied results of 15 cases[1]. Subsequently, in 1952, I reported two more instances of mesenteric thrombosis[2]. To these previously reported cases, I should like to add one more with an unusual etiological background, namely, polyphlebitis.

Case Report

The patient in question is a 37-year old bachelor who was treated at home for a virus infection. Subsequently, after 9 days therapy, he became progressively worse with abdominal pain and phlebitis of the right leg. The patient was hospitalized on 16 December 1957. It was on that date that a surgical consultation was requested. Examination indicated that the patient was acutely ill complaining of abdominal pain plus pain in the right leg. There was a history of vomiting. Nausea was present on occasion but was not a major symptom. Vomiting without nausea on many occasions was noted since the onset of the illness. Abdominal examination revealed slight distention in the left lower quadrant with moderate tenderness over this area. There was no muscle rigidity. A significant physical finding was the fact that there was no tenderness upon superficial palpation of the abdomen. The patient's response to deeper palpation, however, was extreme. This indicated to the examiner that the pathology was confined to a deeper area (the intestine) and had not as yet involved the peritoneal cavity (peritonitis). There was no evidence of shock (B.P. 170/110). The only other significant finding was a right thrombophlebitis which had traveled to the thigh and opposite leg.

A flat plate x-ray of the abdomen showed moderate gaseous distention of the type seen in small bowel obstruction.

A subsequent barium enema revealed no obstruction (Fig. 1). After these x-ray studies were completed, a tentative preoperative diagnosis of mesenteric thrombosis (venous type) was made and the patient was prepared for surgery.

A laparotomy was performed. The peritoneal cavity contained a small amount of bloody fluid. A segment of lower jejunum 9 to 10 inches in length was discolored and pregangrenous. The mesentery to this bowel segment contained venous thrombi without arterial involvement. The pathological picture was clearly one of venous thrombosis. The involved segment was resected including the mesentery with the contained thrombi. An end-to-end anastomosis was performed. The patient had an uneventful recovery and was discharged from the hospital on 31 December 1957. Conservative measures were employed

in the management of the phlebitis which subsided completely during the postoperative period. He has been under the care of his family doctor since his recovery without any unusual complaint. (Patient seen by me on 27 June 1958.)

Comment on Polyphlebitis

In the entire series of cases studied over the years, there was only one other instance of mesenteric venous thrombosis associated with polyphlebitis. This finding was revealed at autopsy.

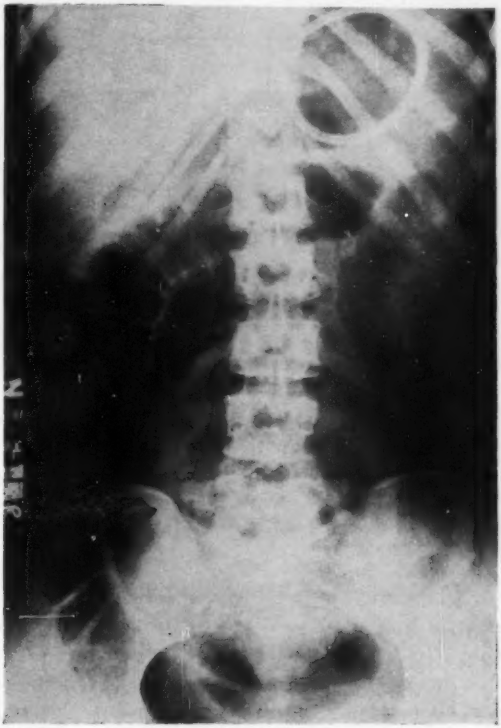

Fig. 1—Scout film of the abdomen showing distended small bowel simulating an intestinal obstruction. A barium enema revealed no obstruction. At surgery mesenteric thrombosis was found.

Migratory polyphlebitis usually involves the surface veins of the legs. It may, however, occur in the deeper veins. Both upper and lower extremities may become the site of this disease. On one occasion I have seen veins on the anterior abdominal wall involved with this process. Migratory polyphlebitis when not associated with *thromboangitis obliterans* (Buerger's disease) should be considered as a forerunner of this disease. Polyphlebitis has been seen in

patients with carcinoma and may be considered as an overt manifestation of a latent neoplasm.

In a discussion on the etiology of this disease, mention should be made of the fact that almost all patients with migratory polyphlebitis present a significant antecedent history. The historical data may be that of trauma to the extremities, frost-bite or an acute systemic febrile disease[3]. In the patient reported here, the antecedent factor was an acute febrile illness interpreted as an acute virus infection. This infection instigated the phlebitis or reawakened it. The migratory nature of the disease found a resting place within the peritoneal cavity producing a venous thrombosis in the jejunal mesentery with its subsequent concatenation of events.

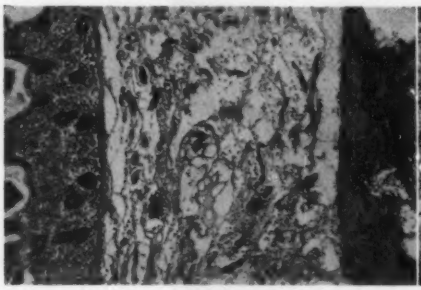

Fig. 2

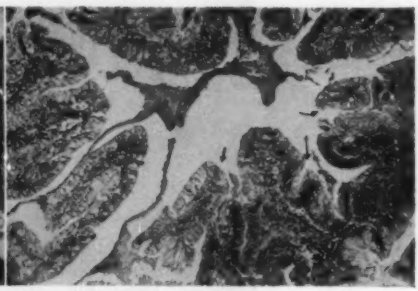

Fig. 3

Fig. 2–Photomicrograph (125x) illustrating the hemorrhagic infarction. Hemorrhage is noted in the submucosa (center of photograph). Arrow marks hemorrhage in muscle layer with disruption in the normal continuity of the muscle bundles.

Fig. 3–Photomicrograph (high power view) showing the disruption of the mucosal surface (marked by arrows) secondary to edema. The lumen contains semicoagulated blood. In the lower right area of the photograph dark round cells are seen indicating hemorrhage beneath the mucosa.

Comment on Mesenteric Thrombosis

Many investigators have attempted to explain mesenteric vascular occlusion and each has postulated his own concept of the pathogenesis. There are two types, arterial and venous. Arterial occlusion may result either from a thrombus or an embolism. Embolism of the superior mesenteric artery may occur in patients with heart disease. The embolus usually arises in the left side of the heart from vegetations on the valves or a thrombus in the auricle. When arterial thrombosis occurs, it may be traced to atheromatous degeneration of the vessel wall. The superior mesenteric artery is more frequently concerned especially in embolism than is the inferior mesenteric artery.

Venous thrombosis of the mesenteric vessels is usually associated with infection in organs or viscera that are tributaries to the portal vein. The conditions usually antedating the thrombosis are appendicitis, pelvic inflammatory diseases, or ulcerating carcinoma. Primary mesenteric venous thrombosis is rare.

It may be a manifestation of migratory phlebitis, or phlebosclerosis. In secondary thrombosis when inflammatory lesions are not in evidence, injury to the vessel wall (as in constrictions from an incarcerated hernia) should be remembered as an etiological factor. A situation of this type is spoken of as autochthonus thrombosis.

The pathological condition resulting from vascular occlusion is infarction. The infarction is usually of the (red) hemorrhagic variety involving the jejunal or ileal segment supplied by the vascular channels occluded.

Macroscopically, the involved segment of intestine has a wall which is thickened, edematous, dark red in color, rapidly becoming gangrenous. The entire lumen contains thick semicoagulated blood. The peritoneal cavity will contain varying amounts of blood-colored fluid depending upon the duration and extent of the pathologic process. Peritonitis is the inevitable complication of long standing vascular occlusion.

Microscopically, the lumen of the bowel is filled with a large amount of hemorrhagic fluid. The mucosal lining shows all stages of degeneration ranging from edema to necrosis. The hemorrhage and edema are seen in the submucosa and muscle layer especially, as demonstrated in Figures 2 and 3.

Clinical Picture

Attempts have been made to outline a clinical symptom complex which might serve as a basis for the early diagnosis of mesenteric occlusion[4,5]. No definite pattern can be evolved because the signs and symptoms depend upon the amount of bowel involved and the presence or absence of complications. The symptomatology can range from a profound shock-like picture to nonspecific generalized abdominal signs and symptoms. Diagnosis of mesenteric thrombosis is most difficult. The presence, however, of migratory polyphlebitis in either the upper or lower extremities with the development of acute abdominal signs and symptoms should suggest to the examiner that mesenteric venous thrombosis is present.

Conclusions

1. An unusual case presentation is made of mesenteric vascular occlusion secondary to migratory polyphlebitis of the leg.

2. A brief discussion is made on the etiology, pathology and clinical picture of this entity.

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PERFORATION OF THE RECTUM FOLLOWING BARIUM ENEMA

ROBERT K. SPIRO, M.D., F.A.C.G.*

Bloomfield, N. J.

Diagnostic procedures are being used with increasing frequency, and with consequent improvement in medical care. It is imperative, however, to recognize that all such examinations occasionally produce trouble for the patient. This report illustrates an unusual complication associated with a very common diagnostic procedure, i.e., perforation of the rectum at the time of barium enema.

E.M., a 70-year old white female was admitted to The Mountainside Hospital via ambulance on 6 June 1957, complaining of some shortness of breath,

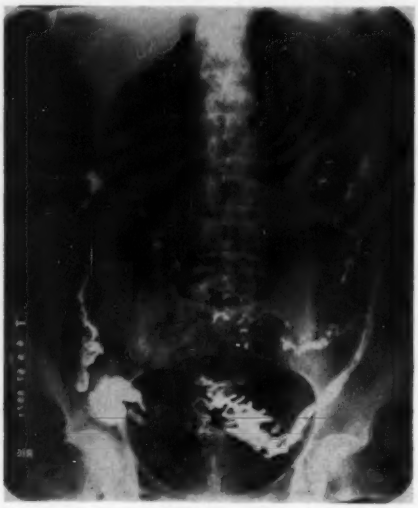

Fig. 1—Considerable air is present in the posterior extraperitoneal soft tissue planes.

fullness of the neck, pain in the rectum, and sensation of bubbles in the subcutaneous tissues of her neck, back, and buttocks.

The patient's health had been good, except for repeated episodes of diarrhea during the past four months. Investigation of the cause of the diarrhea by her family physician so far had been nonproductive. The day of the hospital admission, a barium enema had been performed in the office of a private radiologist.

The radiologist reported that the procedure was accomplished in customary fashion. When the Bardex tube was removed following instillation of barium,

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some blood was noted exuding from the rectum. Suspecting a colon lesion, the radiologist insufflated the colon with air. The patient promptly exhibited signs of shock. Fluroscopic study revealed gas in the posterior extraperitoneal space from the pelvis up to the neck. The insufflation was immediately stopped, the patient made comfortable, and sent to the hospital.

Physical examination at admission revealed a woman in respiratory distress. When placed in Fowler's position, the patient breathed more easily. Crepitus was felt over the full length of the back. A 2 cm. irregular defect was felt 6 cm. above the anus in the right lateral aspect of the rectum. Blood tinged soft barium was in the rectum. The abdomen was soft. Sigmoidoscopic study without insufflation confirmed the rent noted above.

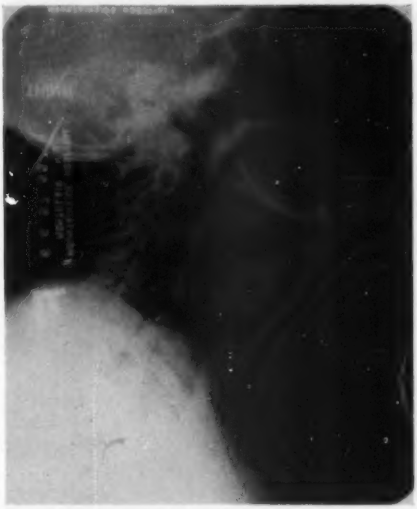

Fig. 2—The air in the soft tissues of the neck indicates the extent of the dissection from point of origin, the perforation of the rectum.

Radiographs (Figures 1 and 2), showed much gas in the soft tissue planes of both neck and abdomen.

Laboratory data revealed a Hgb. of 13.2 gm., R.B.C. 5.02 million, W.B.C., 21.1 thousand with 92 per cent polymorphonuclear cells. The last finding suggested contamination of the extraperitoneal area.

The patient was prepared for surgery and an abdominal exploration done. No signs of intraperitoneal spill were found. The omentum was adherent to the posterior surface of the uterus and the rectosigmoid area, suggestive of a past inflammatory process in that area. No diverticula were evident. A double-

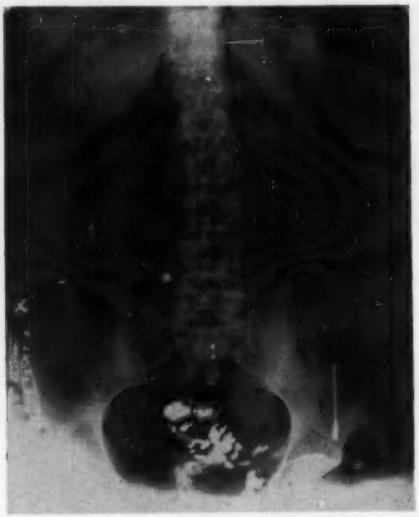

Fig. 3—The decrease in the gas in the abdominal soft tissues is evident, when compared with the earlier abdominal film. See Figure 1.

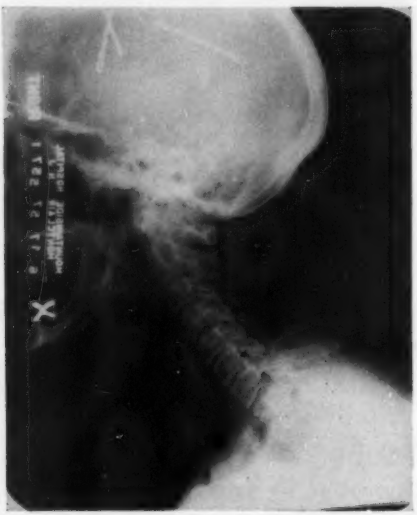

Fig. 4—Considerable reabsorption of the air in the soft tissues of the neck has occurred. Compare with Figure 2.

barreled colostomy was performed in order to completely divert the fecal column away from the perforation site. A rubber drain was placed in the rent, via the rectum, and introduced 4 cm. in the retroperitoneal space to assist drainage and minimize development of a localized abscess. The patient tolerated the procedure well.

Penicillin and streptomycin were administered postoperatively. The colostomy stoma were opened two days postoperatively, and the patient placed on a regular diet.

On 17 June x-rays showed marked absorption of the soft tissue gas (Figs. 3 and 4). The blood count on that day showed a Hgb. of 11.2 gm., a R.B.C. of 3.75 million, and a W.B.C. of 14.3 thousand.

On 18 June 1957, the patient was started on intestinal antibiotics orally, and placed on a liquid diet. On 20 June 1957, continuity of the bowel was restored intraperitoneally by means of a two-layer catgut and silk anastamosis. The patient was placed on a liquid diet on 23 June, and gradually advanced to a full diet. She recuperated well after the second procedure and was discharged well, having normal rectal stools on 30 June 1957. To this date she has continued well.

Comment

The complications of perforation of the rectum frequently are more significant than the perforation itself. Fecal contamination at the locus of injury to the bowel may produce abscess formation in the ischiorectal space, infra- or supralevator areas, or retrorectally, if the trauma be below the peritoneal reflection. The direction of infection can not be predicted.

Supralevator injury may find infection passing retroperitoneally behind the endopelvic and *fascia transversalis*. Retroperitoneal and perinephritic abscess formation may result.

Intraperitoneal perforation of the colon may result in peritonitis and abscess formation in the abdomen itself. In any instance the local abscess must be drained.

In order to reduce fecal contamination to the minimum the primary form of treatment of rectal perforation is a diverting colostomy. In addition, if possible, drainage of such area of perforation may help.

Summary

A case illustrating an unusual complication of barium enema, i.e., perforation of the rectum is presented. The treatment is outlined.

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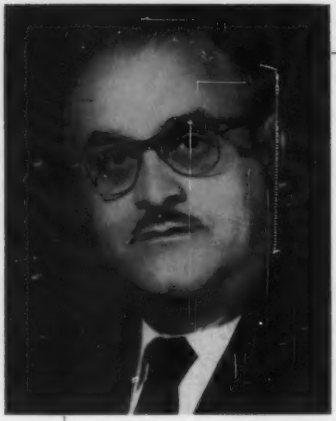

President's Message

Privilege, Honor, Challenge

I believe it is a privilege, as well as, an honor, to be a member of the American College of Gastroenterology. It is especially an honor to serve as an officer, and, I want to take this opportunity to thank the members of the College for electing me their president for the coming year. I shall do all in my power to uphold the dignity and prestige of our organization and hope that each one of you will take some responsibility to further your organization in some little way.

It is a challenge for me, following in the footsteps of those such as Bassler, Upham, Tidmarsh and others, to accept with humility my office of president; to continue in developing the College and further the interests of all those in any way associated in the study of gastrointestinal diseases.

Your Program Committee, under the able direction of Dr. Louis Ochs, Jr. as chairman, arranged an outstanding scientific and social program for the convention, and I hope that many of our members and their guests were able to attend and participate in the instructional and scientific program as well as the nonscientific functions.

To the Woman's Auxiliary, may I also extend my compliments on their outstanding program for the doctor's wives and friends.

All in all, this was an exceptional convention of the American College and I hope you had the satisfaction of having had a part in its success.

Frank J. Borrelli

ABSTRACTS FOR GASTROENTEROLOGISTS

GASTROINTESTINAL TRACT

SYMPTOMATIC MAGNESIUM DEFICIENCY IN MAN: James F. Hammarsten and William O. Smith. New England J. Med. 256:897 (9 May), 1957.

It is well known that magnesium deficiency occurs in man, but it is not generally agreed that it may produce symptoms. A well documented case report is presented of a patient in whom magnesium deficiency with confusion, stupor, tremors, athetoid movements developed after profuse diarrhea and parenteral fluid therapy without added magnesium. The symptoms disappeared with magnesium sulfate therapy, reappeared when this therapy was discontinued and again subsided on reinstitution of the medication.

Recognition of this condition more often by clinicians awaits the development of a simple method of determination. The occurrence of the syndrome described above in patients with a low intake or opportunity of increased loss of magnesium should arouse suspicion and warrant administration of magnesium.

CHESTER S. SVIGALS

ESOPHAGUS

THE PROBLEM OF HIATUS HERNIA COMPLICATED BY PEPTIC ESOPHAGITIS: Gustaf E. Lindskog and John L. Kline. New England J. Med. 257:110 (18 July), 1957.

Twelve cases are presented of peptic esophagitis complicating the sliding type of hiatus hernia. The etiological factor common to all was the reflux of gastric juice through an incompetent esophagogastric sphincter. The symptoms are pain underneath the xiphoid appearing shortly after meals and relieved by antacids. There are often dysphagia and regurgitation, and there may be bleeding.

No single method of treatment suffices for all cases. Surgical correction of the hernia may arrest the early case but when irreversible esophageal stenosis has developed, distal esophagectomy and partial gastrectomy with vagectomy is the procedure of choice. The problem of gastric emptying after vagectomy may be treated by gastrojejunostomy. The authors stress the early repair of sliding hiatus hernias complicated by reflux esophagitis.

ARNOLD STANTON

STOMACH

CONSIDERATIONS ON GASTRODUODENAL ANASTOMOSIS: Pierre Jouanneau. Ann. Chir. 11:1581 (Nov.), 1957.

After studying 75 personal cases in which treatment consisted of partial gastrectomy and gastroduodenal anastomosis, the author refutes certain objections that have been made concerning this type of anastomosis, and claims that cancer of the stomach is not a contraindication.

Emphasis is laid on the necessity of placing the cut end of the stomach horizontally. Several technical points are described.

The results of this type of anastomosis are compared with 96 cases in which partial gastrectomy was terminated by a gastrojejunal anastomosis. Gastroduodenal anastomosis is superior from a threefold point of view: the patient feels more comfortable, digestion is easier and regain of weight is more rapid.

Gastroduodenal anastomosis necessitates a mobile and well vascularized duodenum, and because of these imperatives this procedure can be carried out without risk in only 20 per cent of cases.

A CONSIDERATION OF THE DIAGNOSIS AND TREATMENT OF 132 CASES OF ACUTE GASTRODUODENAL HEMORRHAGE: G. Lagache and G. Soots. Arch. mal. app. dig. 46:1114 (Nov.), 1957.

The authors make a critical study of the diagnosis and treatment of acute gastroduodenal hemorrhages based on 132 cases.

Emergency radiology, carried out 118 times, enabled an etiological diagnosis to be made in 81.3 per cent of the cases, with a proportion of 67.2 per cent of gastroduodenal ulcers.

From the therapeutic point of view, particular attention was paid to the indications in the age of the patient and the recurrence of hemorrhage.

If the diagnosis is conclusive, patients over 50 should, in theory, be operated on at the first hematemesis, whereas in young patients the operation can be delayed until the second or third hemorrhage.

An analysis of the results seems to indicate that a deferred operation, considerably reduces operative fatalities and offers more advantages than the emergency operation.

GUY ALBOT

RADIOLOGICAL STUDY OF PEDUNCULATED AND PROCIDENT TUMORS OF THE STOMACH: Maurice Feldman. Arch. mal. app. dig. 46:1133 (Nov.), 1957

The author studied pedunculated and procident tumors of the stomach so as to give precise information about their frequency and radiological symptoms. Clinical study and anatomopathological tests show that these tumors are relatively rare. Autopsies show 0.076 per cent. He emphasizes in particular the difficulties encountered in the diagnosis of these tumors and discusses their radiological characteristics.

GUY ALBOT

SURGICAL TREATMENT FOR ULCEROUS HEMORRHAGE: M. Mercadier. Arch. mal. app. dig. 46:1088 (Nov.), 1957.

The timing of the operation depends on the effectiveness of the reanimation. The operation can only be immediate (1st day) or retarded (2nd day). It cannot be delayed without risk.

The reanimation, blood transfusion and oxygen therapy, must be strictly adapted to the seriousness of the hemorrhage. If insufficient it will cause a secondary shock and if excessive will cause a relapse due to vascular overloading.

The "complication hemorrhage" of ulcers, defined by gastroenterologists, must without delay be placed in the hands of a competent reanimator. The surgeon should only intervene when reanimation has failed, but the decision to operate should be taken without delay, within the first 48 hours.

GUY ALBOT

INTESTINES

SMALL BOWEL VOLVULUS: Grafton A. Smith and John F. Perry, Jr. Missouri Med. 54:1049 (Nov.), 1957.

The cause of volvulus of the small bowel was: 1. following previous surgery, 79 per cent and 2. those due to congenital anomalies 21 per cent. Formerly the prognosis was grave but early recognition and the use of diagnostic aid of special radiographic technic has reduced the surgical mortality to 13.3 per cent.

VINCENT J. GALANTE

COLONIC POLYPS: REPORT OF THREE CASES: Charles W. Mayo and Martin Y. Laberge. Postgraduate Med. p. 458 (22 Nov.), 1957.

Patients who have had a polyp removed have a 28 per cent chance of having it recur. The authors advise routine examination annually as a follow-up in all these cases.

Three case reports are presented to illustrate the above points. These case reports are followed by a reminder that at postmortem, polyps are found in 2.5 per cent of all cases up to the age of 30. This percentage jumps to 14.2 per cent at age of 50 years or over.

In one month, 176 cases of polyps were found in 1,525 cases sent in for proctological examination at the Mayo Clinic. A polyp may remain benign for 10 to 20 years and then turn malignant. Malignancy usually starts at the tip; the stalk becomes infiltrated later.

Polyps should be removed—even if they are asymptomatic; because they may be precancerous; and because most cancers of the colon start as polypi. After removal, all patients with polypi should undergo annual examination at least once a year.

LIONEL MARKS

JEJUNOGASTRIC INTUSSUSCEPTION FOLLOWING SUBTOTAL GASTRECTOMY: John Van Prohaska, Michael C. Govostis, Herman P. Harms and Shirl O. Evans. Am. J. Surg. 94:776 (Nov.), 1957.

The writers present a review of the literature on this subject matter. The first jejunogastric intussusception was reported in 1917. Since that time there have been 80 similar cases. These included intussusceptions following simple gastrojejunostomies as well as those following subtotal gastrectomies. In 13 cases, the intussusception developed long after the surgical procedure, some as late as two years. The authors note that the only type of intussusception seen following subtotal gastrectomy, is the invagination of the efferent jejunal segment.

The symptoms are given in detail, but epigastric pain and hematemesis were constant in all cases.

Failure to reduce the intussusception by immediate surgical intervention caused death in four cases. In one case, the intussusception was so severe and extensive it required "open" method of reduction through the stomach wall. The patient made a complete recovery.

CARL J. DEPRIZIO

ENTEROPATHOGENIC ESCHERICHIA COLI SEROTYPES: INFECTION OF NEWBORN THROUGH MOTHER: H. W. Oeklitz and E. F. Schmidt. Brit. M. J. 5052:1036 (2 Nov.), 1957.

Fifty-two primary infections were investigated as to cause. 1. Donors of breast milk had pathologic E. coli in stool specimens, 2. milk collected mechanically from mothers was found to be contaminated, 3. those in home delivery had mothers carrying the germ in the stools.

While pathologic E. coli infection is usually infant contact, it can readily be transmitted to adult hosts, nurses, mothers, dietitians and others, even from infant through carrier to infant.

To combat this transmissal more emphasis must be placed on prophylaxis of hospital staff, mothers, physicians and others coming in contact with infants which should include bacteriologic recognition of these pathogens and appropriate therapy.

J. Edward Brown

EIGHT CASES OF TUMOR OF THE SMALL INTESTINE: M. Verhaeghe, J. Devambez, G. Soots and Godefroy-Vendeville. Arch. mal. app. digest. 46:1073-1088 (Nov.), 1957.

The authors give an account of 8 new cases of tumors of the small intestine which brings the total of those studied over 12 years to 10. Seven out of 8 concern malignant tumors, epithelial (2 cases) or conjunctive (5 cases). Each account is given in fullest detail from the point of view of antecedents, clinical and radiological symptoms, methods of treatment, the pathological anatomy and long-term results. These various features are then discussed. Among the antecedents they noted the association with the 2 epitheliomas of the small intestine of another glandular sigmoid epithelioma in one case, and of a malpigian pretragian epithelioma in the other. The tumor was only seen twice; 5 times out of 8 there was an occlusion; twice there was an invagination, only discovered on operation. Radiology has never been able to give accurately the position of the obstruction in the small intestine. Two laparotomies, four segmentary resections of the ileum and two hemicolectomies on the right side were carried out. The best survival record after 2 years, is of a 12-year old child with a reticulosarcoma of the small intestine, the only case in which surgery and radiotherapy have been used in conjunction.

Guy Albot

LIVER AND BILIARY TRACT

NEW EPIDEMIOLOGIC ASPECTS OF HEPATIC DISTOMATOSIS IN THE LYON'S AREA: J. Coudert and F. Triozon. La Presse Medicale 63:1586-1588 (5 Oct.), 1957.

Until now, European distomatosis was observed in France as an exceptional and isolated case, in sheep breeding areas. A recent epidemic aspect (about 500 people) was observed in the southeast area, north of Lyons, in France. In this country the bovine herd exhibits a high rate of parasitism with *Fasciola hepatica*, and the human contaminant was found to be the wild water-cress.

Clinically, symptoms of influenza, or paratyphic infection, or perihepatitis were observed. Numerous cases were uncovered among patients of the same family, who had no clinical symptoms.

High eosinophilic rate in the blood picture is a good alarm symptom. Intradermal test with lyophilized antigen gives early and specific response. The discovery of eggs in stool or bile appears as a valuable test, but too late for diagnostic purpose and more it requires special investigative technics. The treatment by emetine is active in early infestation, but frequently requires two cures.

LIVER SURGERY IN RELATION TO DISEASES OF THE COLON AND RECTUM: Ronald W. Raven. Proc. Roy. Soc. Med. 50:775 (Oct.), 1957.

The treatment of various conditions by surgical means may be greatly influenced by the state of the liver. This is especially so in many colonic and rectal diseases. Behavior of the liver under various circumstances has improved by biophysical and biochemical investigations. In interpreting the results of liver function one must remember that liver parenchyma has a large functional reserve. Approximately 1/10 of normal parenchyma is sufficient to maintain adequate function.

In conservative treatment of ulcerative colitis a group of tests done at intervals

showing evidence of increasing liver damage may influence the decision to carry out surgical treatment before it's too late.

In patients with extensive liver damage as shown by a group of liver tests the necessity for exploratory laparotomy may be avoided. (A positive urinary urobilinogen, retention bromsulfalein and alkaline phosphatase above 13 units).

A. J. BRENNER

SYMPOSIUM ON JAUNDICE I: VIRAL HEPATITIS: J. V. Cable. New Zealand M. J. 56:500 (Oct.), 1957.

The author reviews viral hepatitis under the subject of jaundice from a historical point of view. He states that in infants the anicteric form of viral hepatitis occurs in about 90 per cent of the cases, while in 20 per cent of the cases, adults may have it and it remains anicteric. Another interesting point brought out in the article is that the disease is increasing in severity with increasing years. Mention is also made of the transaminase estimation which may be of great value in the future to determine these anicteric cases.

In relation to the serum hepatitis, it has been shown that one-half per cent of the population carries the virus in their blood without showing any signs or symptoms of the disease.

As to treatment, no notable advances have been made. A good level of nutrition is maintained. The author urges that the patient be in bed as long as acute symptoms persist, after that regardless of the jaundice, they should not be forced to stay in bed more than an hour after each meal. The author also suggests that the patient could be discharged from the hospital after the serum bilirubin has fallen below 1.5 mg. As to diet, approximately 3,000 calories daily containing 150 gm. each of protein and fat is recommended. Cortisone has been suggested in cases of jaundice due to viral hepatitis where progress is slow and may be of help in shortening the course of the disease.

LIONEL MARKS

SYMPOSIUM ON JAUNDICE II: THE PRACTICAL VALUE OF LIVER FUNCTION TESTS AND LIVER BIOPSY: Ian Prior. New Zealand M. J. 56:503 (Oct.), 1957.

The liver has many complex functions and liver function tests may play an accurate part in diagnosis of liver disease when closely correlated with history and physical findings. Liver function tests are used in diagnosis or jaundice; differentiation between obstructive type and parenchymal diseases; assessment of progress and response to treatment, diagnosis of liver disease in absence of jaundice; investigation of systemic disorders involving the liver; assessment of involvement of liver by neoplasms.

The selection of tests are urine analysis, feces, serum bilirubin, bromsulfalein dye excretion, serum alkaline phosphatase and liver biopsy. The indications for liver biopsy are discussed.

LIONEL MARKS

SYMPOSIUM ON JAUNDICE III: THE PHYSIOLOGICAL BASIS OF SOME LIVER FUNCTION TESTS: J. D. Reid. New Zealand M. J. 56:507 (Oct.), 1957.

The author discusses the basic established physiological known facts in liver function tests as well as some of the nonestablished newer observations.

One of the more recent tests is the measurement of G-O transaminase in parenchymal liver damage. The author thinks this is undoubtedly due to the liberation of G-O transaminase by the destroyed liver cells. Also a newer test is the chromatographic analysis of the urine. This will show an increase in tyrosine and cystine in the urine in acute hepatitis. The measurement of gamma globulin also rises in parenchymal diseases of the liver while the albumin falls. Most of the flocculation tests depend upon a balance between the serum albumin and serum globulin. Alkaline phosphatase is increased in production in the liver after ligation of the bile ducts. The above principles, some of which are well established and some of which are still experimental, form a physiological basis for the more common liver function tests.

LIONEL MARKS

SYMPOSIUM ON JAUNDICE IV: JAUNDICE IN THE NEWBORN AND ITS MANAGEMENT—MEDICAL ASPECTS: J. M. Watt. New Zealand M. J. 56:512 (Oct.), 1957.

The author discusses the physiology of the liver at birth and the significance of jaundice in the newborn period. One point is particularly brought out, that it is difficult to decide whether jaundice in the newborn is of significance.

Jaundice appearing in the first 24 hours of life must be regarded as abnormal. An increase in jaundice after the first week is likely pathological. Jaundice persisting after the third week is definitely abnormal and requires investigation. Hemolytic disease in the newborn is due to Rh incompatibility. Another important point in the newborn that develops jaundice is due to atresia of the bile ducts. The final group in the neonatal is due to infection; this may be of various types, such as virus and syphilis.

The treatment varies as to cause. The great majority of physiological jaundice requires no treatment. The treatment of jaundice secondary to infection follows the usual lines of antibiotic therapy. Jaundice then in the newborn is due to physiological immaturity of the liver; excessive hemolysis; infection. There are still large gaps in our knowledge which will have to be filled out with more research.

LIONEL MARKS

SYMPOSIUM ON JAUNDICE V: VIRAL HEPATITIS WITH MANIFESTATIONS SUGGESTING BILE DUCT OBSTRUCTION: J. D. Reid. New Zealand M. J. 56:518 (Oct.), 1957.

It is important to differentiate between viral hepatitis and jaundice due to biliary obstruction. Some forms of viral hepatitis show no significant changes in laboratory tests from those seen in many cases of biliary calculi or tumor. Surgery in viral hepatitis has a higher mortality than in other types of biliary surgery. At the moment there is no special test for viral hepatitis. Recently it has been shown that there occurs in hepatitis a rise in serum iron levels; serum transaminase levels may distinguish between jaundice due to hepatitis and extrahepatic obstruction.

Needle biopsy is of considerable value when it is positive. In the middle-aged and elderly, differentiation between obstruction and disease of the liver parenchyma is difficult. The author states that in view of the uncertainty of the underlying pathology, the condition should be referred to as hepatitis with obstruction rather than as cholangiolitic hepatitis.

LIONEL MARKS

INTRAVENOUS CHOLANGIOGRAPHY AND CHOLECYSTOGRAPHY: Curtis H. Burge. Texas J. Med. 53:843 (Nov.), 1957.

This paper reviews an extensive experience of 150 cases of intravenous cholangiography and cholecystography. It is the author's conclusion that these are valuable procedures when indicated and are no more prone to produce undesirable side-effects than intravenous urography. In this series of 150 cases no serious side-effects were detected. The author feels that the intravenous procedures should not be used when an oral cholecystogram might give the same information. This procedure is used to best advantage in patients with nonvisualization of the gallbladder by the oral method and in patients with previous cholecystectomy or having symptoms referrable to the biliary tract. This method may be used in those patients with severe vomiting, diarrhea and other abnormalities which prevent satisfactory oral cholecystograms. The author points out that intravenous cholangiography may frequently differentiate between obstructive and nonobstructive jaundice. Examination of the common duct showing stone dilation or stricture may be invaluable following cholecystectomy. Finally, the author points out that tomography is of great value in doubtful examinations in which there is at least some concentration of the material in the biliary ducts or

gallbladders. In this author's experience paregoric is apparently used as a matter of convenience although 7½ mg. of morphine may be used to produce contraction of the sphincter of Oddi routinely in these patients.

RALPH D. EICHHORN

INTRAOPERATIVE MANOMETRIC STUDY OF THE BILE DUCTS: A. Fritsch. J. Internat. Coll. Surgeons 28:557 (Nov.), 1957.

Intraoperative manometric studies of the gallbladder and common bile duct were found to be of little practical value as an isolated study to determine pathology of the organs involved. Greater diagnostic importance was attributed to cholangiography.

Other objections relative to gallbladder manometric studies are cited, which convinces the author of the relative practical uselessness of these procedures.

EZRA J. EPSTEIN

PANCREAS

ACUTE PANCREATIC LESIONS IN PATIENTS TREATED WITH ACTH AND ADRENAL CORTICOIDS: Frank A. Carone and Averill A. Liebow. New England J. Med. 257:690 (10 Oct.), 1957.

Pancreatic necrosis has been noted in experimental rabbits treated with corticosteroids. Paradoxically, ACTH has been used clinically in the treatment of acute pancreatitis.

Investigation by the authors revealed that in 16 of the 54 patients treated with ACTH or adrenocortical steroids; on autopsy, there was histological evidence of acute pancreatitis or fat necrosis. In the control series of the 54 patients, there were two cases of acute pancreatitis without fat necrosis. Ectasia of pancreatic acinar occurred in 32 of the 54 patients in the steroid treated group and in 13 of the control series. These acinar changes were similar to those occurring in the cortisone treated rabbit and also to those described in patients with uremia, ulcerative colitis and high intestinal obstruction.

Although short term therapy with ACTH has been stated to be beneficial in the treatment of acute pancreatitis, caution should be employed in the prolonged use of these agents on the basis of evidence of injury to the pancreas presented here.

H. M. ROBINSON

GASTRIC HYPERSECRETION, PEPTIC ULCERATION AND ISLET-CELL TUMOR OF THE PANCREAS (THE ZOLLINGER-ELLISON SYNDROME): Robert M. Donaldson, Jr., Paul R. von Eigen and Richard W. Dwight. New England J. Med. 257:965 (14 Nov.), 1957.

This case report concerns the findings in the laboratory of noninsulin-producing islet-cell tumors of the pancreas and multiple jejunal ulcerations, one of which had perforated.

An autopsy revealed a grossly normal pancreas, except for the presence of two tumor masses. Microscopically, these tumors were found to be islet-cell carcinomas. One peripancreatic lymp node contained islet-cell carcinoma.

Previously, 21 cases in which there appears to have been an association between noninsulin islet-cells, tumors of the pancreas and peptic ulceration have been reported. These cases were thought to be coincidental and suggested that there might be an etiologic relation between the islet-cell tumor and the occurrence of peptic ulcer. More recently, Ellison collected 24 cases from various sources and postulated the existence of a clinical entity consisting of gastric hypersecretion and hyperacidity, recurrent and often atypically located peptic ulceration and the presence of noninsulin-producing islet-cell tumors of the pancreas. It has been suggested that this entity be called the Zollinger-Ellison syndrome.

JOHN E. COX

STEROID THERAPY IN ACUTE PANCREATITIS: Murrel H. Kaplan. Am. J. Digest. Dis. 2:696-702 (Dec.), 1957.

The outlook in surgically treated acute pancreatitis has recently been materially improved by the use of cortisone. The reported cases are still too few to be statistically significant, but when it is recalled that they represent the so-called malignant type of disease, in which there admittedly is little or no chance of survival, these isolated cases become of great significance. Cortisone has also been used successfully in the nonsurgical management of the malignant type of pancreatitis. Patients with this type of disease have little chance of surviving, no matter how they are treated, in contrast to those with the benign type in which survival occurs with the usual conservative measures.

WALTER CANE

PATHOLOGY AND LABORATORY RESEARCH

EFFECT OF VARYING DOSES OF RESERPINE ON GASTRIC SECRETION: Daniel Liebowitz and John V. Carbone. New England J. Med. 257:227 (1 Aug.), 1957.

Reserpine has been shown to increase gastric acidity in both man and animals. Therefore, a group of normal persons was studied after intake of various doses of this drug. The gastric analysis was performed and blood pepsinogen levels as well as 24-hour uropepsin excretion were determined. A significant rise of hydrochloric acid occurred after a daily dosage of 0.32 mg. as well as 0.5 mg. daily, while no change was noticed with small dosage (0.16 mg. daily). Uropepsin, blood pepsinogen, and viscosity of the gastric juice remained unchanged during the intake of Reserpine. A hypothalamic stimulation by large doses of the drug seems to be responsible for the increase of gastric acidity.

H. B. EISENSTADT

DETERMINATION OF PANCREATIC LIPASE IN HUMAN SMALL INTESTINAL CONTENT: B. Borgstrom. Scandinav. J. Clin. & Lab. Invest. 9:226 (Sept.), 1957.

Two methods for assay of pancreatic lipase based on the rate of clearance of an artificial amount of Triolein emulsion have been proposed. The rate of clearance of the emulsion is proportional to the amount of lipase present in the small intestine. One method, which eliminates the effect of the bile constituents by precipitating the proteins from the intestinal substances with ethanol, has been found to be the most accurate and reliable method and is described in detail.

EZRA J. EPSTEIN

DETERMINATION OF TRYPSIN AND CHYMOTRYPSIN IN HUMAN INTESTINAL CONTENT: G. Lundh. Scandinav. J. Clin. & Lab. Invest. 9:229 (Sept.), 1957.

An improvement over the older methods which determine proteolytic activity of duodenal and pancreatic juices has been devised. With a modification of Schwert and Takenaka's (1955) spectrophotometric method for enzyme determination, it is now possible to test for specific tryptic and chymotryptic activity of the intestinal juices rather than for the less specific proteolytic activity of the whole intestinal content.

By treating intestinal juices with ethyl alcohol, the interfering substances of intestinal contents can be precipitated out and the resultant solution contains only trypsin and chymotrypsin. This has been proven by comparing the optical curves caused by these solutions with the curves produced by known quantities of trypsin and chymotrypsin.

EZRA J. EPSTEIN

THE SIGNIFICANCE OF ALTERATIONS IN SERUM ENZYMES IN THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE: Felix Wroblewski. A.M.A. Arch. Int. Med. 100:635 (Oct.), 1957.

The author believes that the concomitant determination of alkaline phosphatase, glutamic oxalacetic and glutamic pyruvic transaminases will clarify the majority of cases of jaundice. Obstructive surgical jaundice is usually associated with an alkaline phosphatase of greater than 10 units, while parenchymal jaundice has a value of less than 10 units. SGP transaminase shows greater increase than SGO transaminase in obstructive jaundice with levels less than 400 units. Most types of medical jaundice show more SGO transaminase increase than SGP except acute hepatitis that produces enzyme changes similar to obstructive jaundice. The values in infectious hepatitis, however, exceed 500 units. Toxic hepatitis as seen with Thorazine administration will show alterations just as obstructive jaundice, but the transaminases fall to normal at once if the drug is withheld. In contrast, bilirubin and alkaline phosphatase will show persistent elevation, under those circumstances; they might even increase in spite of withdrawal of the drug.

H. B. EISENSTADT

CHOLECYSTOGRAPHIC MEASUREMENT OF BILIARY DYSTONIAS: Pierre Pizon. La Presse Medicale 65:1588-1591 (5 Oct.), 1957.

With the patient lying on the stomach we proceed with an x-ray of the gallbladder, once it has been rendered opaque by the ingestion of a cholecystographic sustance. Evacuation is then provoked using a medicine combining lecithin, saccharine, glycerine, old rum, and methyl and propyl paroxybenzoate. The patient is placed on the stomach and x-rays are taken at the most favorable moments; that is to say 8, 15, 25, 35, and 45 minutes later, with the operator being careful that the patient does not move the slightest during the 45 minutes. Since the patient remains in the same position we are assured of having identical x-rays.

The biliary surfaces are measured by means of decalcomanias of calibrated paper. The x-ray of the first lumbar vertebra is joined to this decalcomania and is considered as a frame of reference. The size of the gallbladder at rest is expressed as a proportion of the vertebral surface. It is, on an average, 108 per cent the size of the first vertebra with extremes of 52 and 164 per cent. Following the time intervals of the surfaces calculated during evacuation compared to the original bladder surface, we can establish the representative curve of evacuation.

Normal evacuation of an orthotonic and orthokinetic gallbladder is expressed by a regular exponential curve with the residual surface, after 45 minutes, 26 to 65 per cent of the original surface.

A moderately hypertonic but orthokinetic gallbladder empties regularly following a rectalinear curve; the residual surface is normal between 26 to 65 per cent.

A dyskinetic bladder empties following an "S" curve as a result of irregularities which develop before a lapse of 20 minutes. There will certainly be a hypertonia if the residual surface is below 26 per cent. The hypertonia is only probable if the residual surface is between 26 and 34 per cent.

A dyskinetic and hypertonic bladder also presents a degressive "S" curve but the irregularities are noticed after 15 minutes. Depending upon the slope of the curve and the residual surface we are able to distinguish a hypotonic (final surface greater than 65 per cent), an asystolic (final surface greater than 85 per cent), and finally an akinetic atony.

We always complete the examination by studying the biliary ducts, and if we find it necessary to study the common bile duct we must pay close attention to the Lutkens sphincter whose abnormalities seem to paralyze the bladder more quickly and more completely than abnormalities of the Oddi sphincter.

In conclusion, a study of the variations of the evacuation surface of the gallbladder and the establishment of a representative curve, constitute a delicate analysis of biliary dystonias.

BOOK REVIEW FOR GASTROENTEROLOGISTS

CLINICAL PROCTOLOGY: J. Peerman Nesselrod, B.S., M.S., M.Sc. (Med.), M.D., F.A.C.S., F.A.P.S., Assistant Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Division of Proctology, Evanston Hospital, Evanston, Ill.; Diplomate of the American Board of Proctology; Captain (M.C.) U.S.N.R. Second Edition. 296 pages, illustrated. W. B. Saunders Company, Philadelphia, Pa., 1957. Price $7.00.

A concise and comprehensive text dealing with proctological diagnosis and therapy. The author has given the general practitioner a small, but working volume, which should be of great help in diagnosis and office management of rectal complaints.

On page 59 the author calls attention to proctologic examinations in relation to other fields, medicine, pediatrics, geriatrics, urology, gynecology, general surgery and orthopedics and this should be read carefully by the physician. On page 83—palliative treatment, the author advises bed rest and ice packs which is contrary to the old teaching of hot baths and/or hot applications. Later, moist heat in the form of hot wet packs and sitz baths can be substituted for the cold packs. On pages 84-88, injection versus surgery of hemorrhoids, is well presented.

On page 105, anal fissure, etiology, various stages, pathology, etc. and treatment, injection and/or surgery, are also well presented. On pages 204 and 205 in endoscopic views in color and black and white, an attempt is made to show the normal and abnormal findings. Unfortunately, they are not the best, although this should not detract from the rest of the text.

Neoplastic diseases, anal pruritus and miscellaneous disorders complete "Clinical Proctology".

References at the end of the chapters and a cross index, plus clear type and illustrations add to the attractive monograph.

For everyday reference, it is undoubtedly a worthwhile addition to the physician's library.

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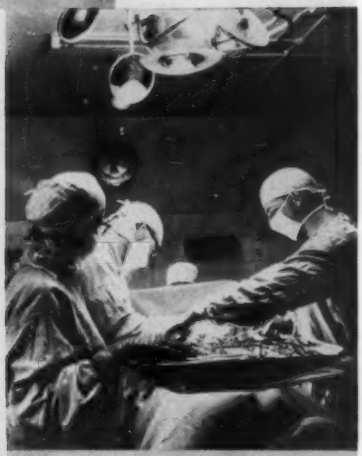

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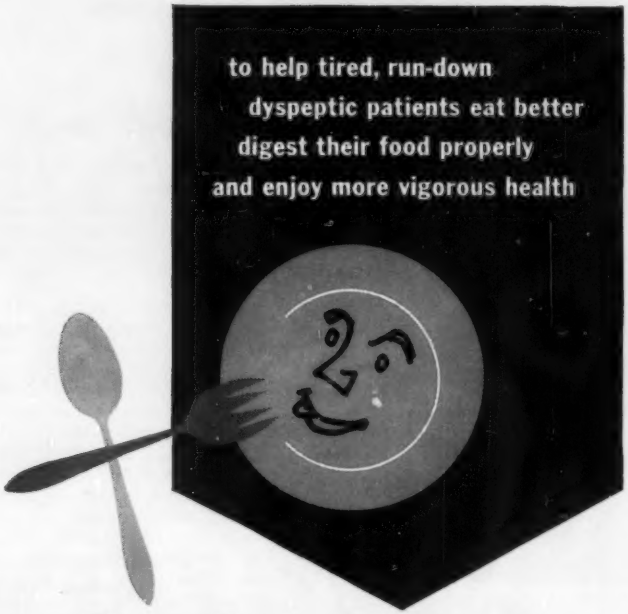

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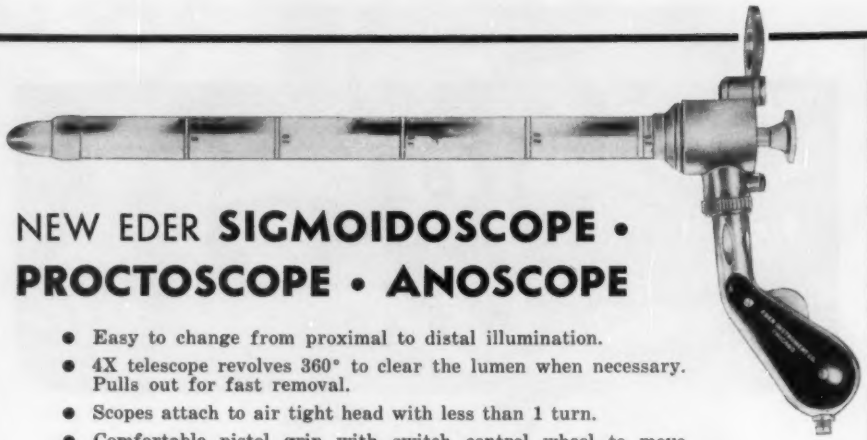

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[1] *American Journal of Gastroenterology* 28:439, 1957.

[2] *British Medical Journal* 2:827, 1955.

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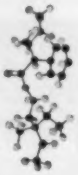
three-way mechanism of action  in one molecule

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1. Berndt, R.: Arzneimittel-Forsch. 5:711 (Dec.) 1955.
2. Peiser, U.: Med. Klin. 50:1479 (Sept. 2) 1955.
3. Winter, H.: Medizinische, p. 1206 (Aug. 27) 1955.

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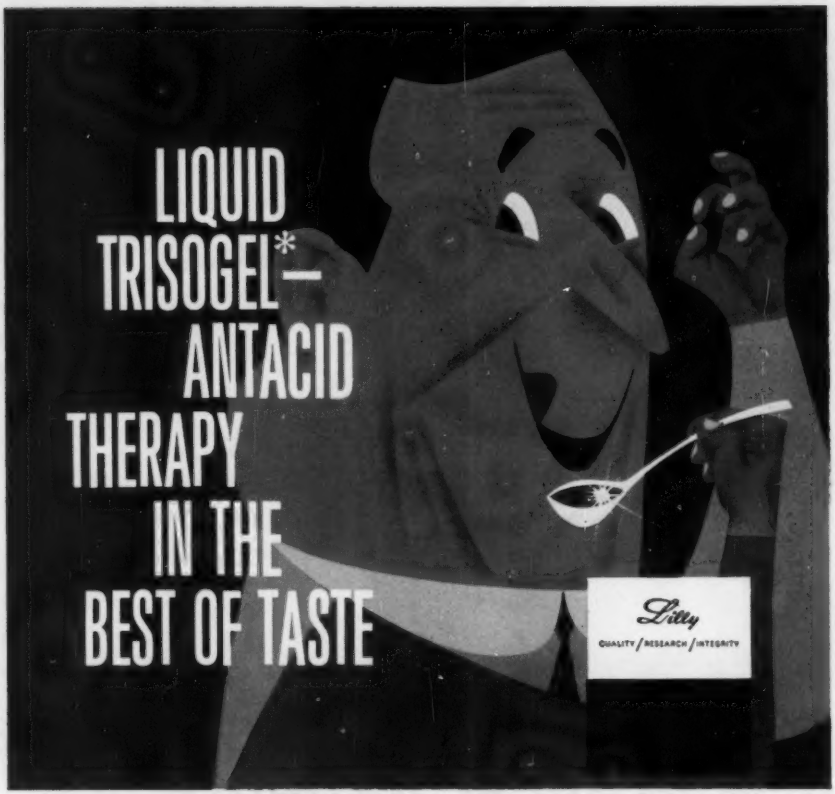

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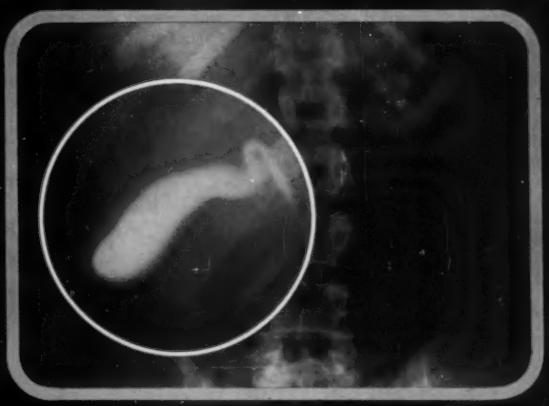

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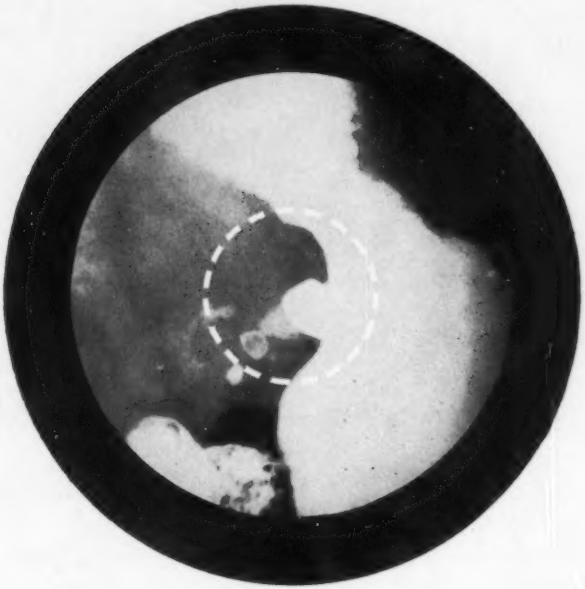

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